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ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY ARISING FROM THE USE OF ASBESTOS IN ONTARIO

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180 Dundas Street
Toronto, Ontario
Tuesday,
June 30, 1981

VOLUME XV

ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

ARISING FROM THE USE OF ASBESTOS IN ONTARIO

VOLUME XV

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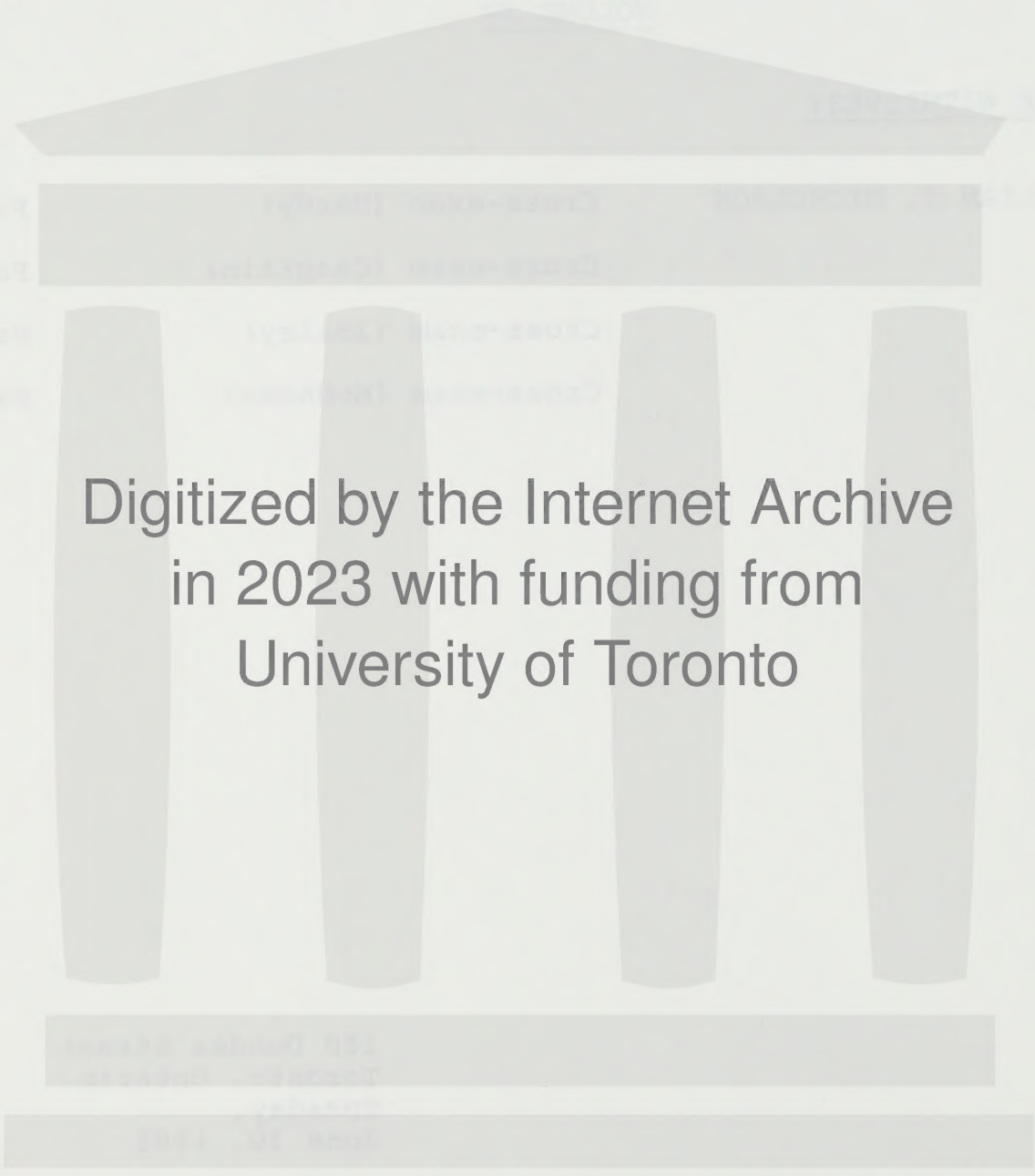
180 Dundas Street
Toronto, Ontario
Tuesday,
June 30, 1981



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VOLUME XV

THE FURTHER PROCEEDINGS OF THIS
INQUIRY RESUMED PURSUANT TO ADJOURNMENT

APPEARANCES AS HERETOFORE NOTED

DR. WILLIAM J. NICHOLSON, PREVIOUSLY AFFIRMED, RESUMES THE STAND

DR. DUPRE: Good morning, class. Who is number one in the batting lineup?

MR. HARDY: I am, Mr. Chairman.

DR. DUPRE: Mr. Hardy, you are going to lead off?

MR. HARDY: Yes, sir.

DR. DUPRE: Very well. Proceed, please.

CROSS-EXAMINATION BY MR. HARDY

Q. Yesterday you were asked a question, Dr. Nicholson, about monitoring schools. I believe you indicated you did not believe it was very useful to do air sampling in schools, is that correct?

A. To base corrective action upon an air sampling program.

Q. Maybe you could just repeat why it is you don't believe that's a valuable tool?

A. Because if there are damaged surfaces, or

A. (cont'd.) you can see visually that asbestos has in the past been disrupted by the kids, or is physically deteriorating, the decision should be based upon that fact, because if it has taken place in the past, it can take place in the future, and one need not do air sampling to verify that there has been and probably will be a problem.

Additionally, air sampling can be misleading in that it can only tell you what is the condition at that one particular point in time, which may not reflect conditions at other times where it would be of concern. It would be difficult to catch the disruption directly with an air sampling program, that would reveal the full scope of the problem.

There can be reasons for doing air sampling to...but in general, for control purposes, it is unnecessary.

Q. I gather air sampling, as you see it, might miss measurements during disruption of the material on the ceiling?

A. That's correct.

Q. I gather those measurements, were they taken, would be higher than what would be found with average sampling during the day?

A. If you sampled enough times that you picked them up, you could do a, in essence, year-weighted average that would reflect the total conditions, but that's not a practical course of action.

Q. I gather you believe that it's important in terms of medical consequences, there are those sorts of exposures at some time during the day?

A. The higher the exposure, the greater the consequence, that's correct. Whether it's...you know, whether it would be the exposure...it isn't so much...it's the total dose that would be the most important. I mean, you could have a high exposure for a few minutes that would have less consequence

A. (cont'd.) than an average exposure over a day or so, at much lower levels. It would depend upon the... the health consequences, I think, depend upon the total amount of asbestos inhaled.

Q. You would be worried that by doing air sampling you might get a low average that would not pick up the peak exposure during destruction, and thus lead to an incorrect conclusion about whether...

A. No, just that the air sampling has no...to try to get an average that would reflect every condition is just not a practical sort of thing. In each individual school you would be having every analytical instrument in Canada involved in trying to make that assessment.

Even when you did so, you would have less information that a person with common sense can tell by looking at the material.

Q. So in other words, it would be your view that if common sense shows you on the ceiling that the asbestos has been poked, that's enough information to make a decision to take corrective action?

A. In general, that's correct. We are talking about issues that have a spectrum of problems, a spectrum of severity of problems. It might be one simple little corner that has a water leak, or something like that, versus a building where the asbestos is literally just falling down like the moss in the Louisanna swamps...which there is a picture in my book that shows that.

Q. That's certainly not very common in schools, is it?

A. It's fortunately not common, but it's unfortunately common enough that the school problem is a real one.

Q. Yesterday you discussed some occupational standards which have been established in other countries in

Q. (cont'd.) previous years. I believe you indicated that the 1968 standard established in the United Kingdom was based solely on protecting against asbestosis, is that correct?

A. That's correct, and that's how it was stated in their publication in 1968, in the British Occupational Hygiene study.

Q. I believe, did you imply the same was true for the two fiber standard in the United States?

A. The British standard as initially published was, had a certain flexibility to it. It wasn't directly a two fiber standard. It had that as its goal.

The U.S. standard took that information and gave great credence to it, and that's I believe the words, in the establishment of their standard, in the 1972 standard that was set, for the criteria document that preceeded that 1972 and subsequently the 1976 standard.

Q. But that standard-setting criteria document in the United States was not based solely on protecting against asbestosis?

A. Largely, it was. They were certainly not protecting against cancer, and they did not claim to do so.

Q. Well, maybe I could read to you from that criteria document, from the National Institute of Occupational Safety and Health.

A. Which one are you reading from?

Q. From the one that is dated 1972.

A. Okay.

Q. Which is cited, I believe, in your tab nine.

A. Right.

Q. On page Roman numeral one dash one of that document, the first sentences in the report say: "The National Institute for Occupational Safety

Q. (cont'd.) "and Health, NIOSH, recommended that worker exposure to asbestos dust in the workplace be controlled by requiring compliance with the following sections:

Control of worker exposure to the limit stated will prevent asbestosis and more adequately guard against asbestos-induced neoplasms"...

A. Of course.

Q. Which I assume they mean cancer?

A. If you reduce it, you will more adequately guard against it than the ridiculous concentrations that were extant in the industry previously, but that's certainly not protecting against cancer to the extent that you eliminate it in the work force, and that standard certainly doesn't do that and they don't claim to do that in that criteria document.

NIOSH, in fact, in their later criteria document, recommended a standard of zero point one fibers per millilitre, and even admitted that might not protect against cancer.

Q. Isn't it true, Dr. Nicholson, that given your belief that you could extrapolate risk of cancer linearly down to zero, that no standard other than a standard of absolutely no exposure would completely protect against cancer?

A. The only standard that one in fact can say would be absent of risk, is a standard of zero. But it also is recognized that that's, with the asbestos that is in place, is not a totally realizable standard. So the next best thing that you can do is to get as close to that as possible, but in fact the only standard that is absolutely safe for everybody in the country, and one that you could assure people will not produce a single case of cancer, is zero.

Q. That would, similarly that would be true for many constituents in foods - like aflatoxins, which

Q. (cont'd.) also are known to be associated with cancer, and thus the only way to be totally safe, as you talk about linear extrapolation, would be to ban all aflatoxins from foods?

A. To be absolutely safe, that's correct.

Q. Which would mean banning all use of peanuts in foods?

A. If you set as your criterion the goal of absolute safety. Standard setting does not set that goal. In both foods and in, unfortunately, in the occupational setting, they sometimes allow the economics to totally dominate, but they clearly have to take into account feasibility. That's established in the United States enabling legislation of OSHA, to the extent feasible, workers shall be protected from harm throughout their working life.

That document did not call for the absolute protection, and thus we have standards in the workplace that do not afford absolute protection. We have standards in the food industry that do not afford absolute protection, although for added carcinogens to foods, they do. The Delaney Clause is a prohibitive clause: You shall not add a material to foods that has been shown to produce cancer in appropriate animal studies, at any concentration.

Aflatoxin is allowed in food because it is not an added contaminant. It is a mycotoxin from fungus growth through poor storage. Unfortunately, the levels that they allow there are considerable and there are undoubtedly cases of liver cancer that are being produced by it, I'm sure, in our country.

Q. So you believe that the current Food and Drug Administration standard for aflatoxin is too high?

A. I would believe it is too high to protect people fully. It was a standard that was established largely

A. (cont'd.) balancing economics and disease, in a very crude way. But it is not a totally protective standard, as is the standard to prohibit the introduction of red dye number two.

Q. Do I hear you saying that it was a mistake, in considering the aflatoxin, to...and I'm asking this generically because I think it's relevant to asbestos, too...are you saying it was a mistake to consider economic factors in setting the standard for aflatoxin?

A. No, I do not. I'm not making the argument here that one has to achieve an absolute...a standard of absolute safety.

When you look at the situation, as in the case with aflatoxin, if you eliminate it from corn, to eliminate it from peanuts, would be such a disruptive situation that it would, in addition to being literally impractical, but would cause more harm in terms of malnutrition, perhaps death, than its inclusion in there. I mean you can make lots of risk/benefit analyses which are as uncertain as they can be, but nevertheless there is the need in circumstances to balance competing factors.

The wrong way to do it, in some circumstances, is to assign a dollar cost to a life and say we are going to allow so much death because we want so many dollars.

Q. That's not what the Food and Drug Administration did with aflatoxin, is it?

A. I'm not saying...in the background of some of the risk/benefit analyses, that is the process that is going on. If you set a cutoff at whatever the number is, you have to reject so much peanuts for consumption. If you set it higher, you do not have to reject for use so much food. You do not have to spend as much money for proper control, proper storage, control storage facilties, and so on. And the same obtains in the workplace, as we see.

A. (cont'd.) The vinyl chloride standard went to one part per million based upon the recognition that that level was not absolutely safe, but it was very close to being safe and it was something that was achievable with technology, and it turned out it was achievable at a cost to industry that was trivial. In fact, they were making money from the technology that they developed to implement that standard, as they sold it to other companies.

So you are in a very dynamic situation when you are setting standards, and that dynamic situation has to be recognized, and it's recognized in virtually all of the enabling legislation, with the exception of the Delaney Clause. There, largely, you are dealing with things that have very little relevance to significant benefits. The use of red dye number two, is an example. It wouldn't make the darndest bit of difference to the population whether that's in food or out, in terms of the benefits that are derived to them. It has some benefit to the food industry because it makes something look a little prettier, but it certainly doesn't add any nutritive value, and thus the exclusion of that on an absolute basis makes sense.

The exclusion of aflatoxin on an absolute basis does not, because its elimination would be virtually impossible from vast stores in the United States.

Q. We've probably got a little sidetracked, but the way we got into this was by discussing whether the 1972 NIOSH criteria document considered the cancer risk.

A. It did not seriously consider the cancer risk in the levels that it set at that time. Those levels do not protect against cancer. NIOSH recognizes they do not protect against cancer. They were largely chosen because those were the levels that were achievable by industry at that time, and firstly they set a standard of five fibers rather than two fibers, for the period 1972 through 1976. That was simply

A. (cont'd.) because it was felt that that would be possible. They did not think at that time that two fibers would be possible, but said that within five years it would be, and so we will then go to two fibers in five years. By that time the industry would have the technology available to control to that level.

So again it was a dynamic process recognizing both the engineering realities, to some extent the economic realities, and also recognizing the health realities that that would not necessarily protect against cancer, as was manifest very clearly in the second criteria document that was published in 1975.

Q. But they did consider the cancer risk of asbestos in drafting this document? That's what it states.

A. It didn't say that. It simply said that it would reduce the cancer risk from what it was before, where you uncontrolled levels...

Q. Well, I read that to say...

A. That's exactly the statement that you read.

Q. And you don't read that to say that cancer was in their minds when they drafted this document, as well as asbestosis?

A. Certainly cancer can be in their minds, but they weren't setting a standard to control, to eliminate cancer. It wasn't...in fact, if you can class that document to that of vinyl chloride, where they were in fact recognizing only, solely, virtually only the carcinogenic properties, they were making a major consideration that we are going to the lowest level measurable, because the only level that could protect against cancer is zero, and we can measure this as one part per million. They recognize that there is going to be variations about that and there will be excursions above it, but in essence they were setting a zero level within the level of

A. (cont'd.) technology. It was kind of a mumbo-jumbo sort of thing, but that's what they stated they were doing. That's also what they were stated to be doing when they established the criteria of zero point one. That wasn't a technological standard in terms of engineering controls. That was a technological standard in terms of measurement. It's what they would measure as zero, which would be a standard that would consider cancer.

Q. Let's talk a little about only a zero level protecting against cancer. I believe yesterday you discussed a number of studies and from those studies predicted the risk of exposure to one fiber per millilitre per year, and I believe you indicated on a number of occasions yesterday that it is your view that the risk predicted by those studies could be extrapolated to very low levels of exposure. Is that correct?

A. It's the only extrapolation we have. There are no other data that indicate that another extrapolation is appropriate.

Q. But on the other hand, there are also no data at low levels documenting the dose-response relationship at those low levels?

A. At the levels considerably below one fiber per millilitre, that is correct.

Q. Therefore assumptions have to be made about what the dose-response curve does below that point on the curve where we have actual documented data?

A. Yes.

Q. I would just be curious to get your view of a statement made before this Commission on December 12, 1980, when Dr. ...

MISS JOLLEY: Perhaps Dr. Nicholson should have the advantage of having that before him, in order to be able to respond, since that certainly was of concern last week.

DR. DUPRE: Yes.

Was it a long segment?

MR. HARDY: No, it's only one sentence.

DR. DUPRE: If you want to have a look at it and see it in context, perhaps you can just briefly read the part. I'll be happy to have...

MR. HARDY: Certainly, certainly.

Dr. David Sackett, professor of epidemiology and biostatistics at McMaster University, appeared before this Commission and gave a general review of what he called diagnostic tests for causation, and talked about a number of issues, an interpretation of epidemiology, for the benefit of this Commission.

One of the things Dr. Sackett said is the following: "The mathematical properties of shapes and locations are complex, but a few simple rules will assist their interpretation by anyone:

1. Beware of extrapolations beyond actual observations, for they represent acts of faith rather than acts of observation."

DR. DUPRE: For the record, that is, of course, from the proceedings of public meeting number two.

Could you give us the page number, please?

MR. HARDY: Certainly. It's page eight of the prepared address by Dr. Sackett, which is appendix B to the proceedings on December 12, 1980.

DR. DUPRE: Would you like to have that thing in front of you?

THE WITNESS: No.

To a certain extent they are acts of faith. To another extent they are an act of prudence. The alternative, that of not making extrapolations, not making estimates based

THE WITNESS: (cont'd.) upon the best available evidence, would then say that it is perfectly acceptable to allow large populations to be exposed to considerable quantities of asbestos, and not do anything until we see the bodies in the street. I think that's morally reprehensible, and that's the alternative that you are suggesting as being an option to extrapolation and making judgements based upon the best evidence that we have at this time.

MR. HARDY: Q. I don't think I suggested an alternative. I'm just trying to understand what it is that you are assuming, Dr. Nicholson, and that's why I am asking the question.

It's possible though, since we don't have documented data at very low levels, that the dose-response relationship is curvilinear, and would thus predict less risk than a linear extrapolation would predict?

A. It's possible that it is curvilinear and would predict less risk. It's possible it has a different curvilinear shape and would, in fact there could be greater risk at lower levels. We don't know. It could go either way.

Q. Yesterday you talked about immunology as being relevant, perhaps, to the effect of asbestos on the body. As I interpreted that, you were saying that there may be immunological effects which fight against asbestos in the body, which perhaps are more effective at much lower doses.

A. No, no. I didn't say anything about doses. I said that immunological status can bear upon the development of cancer that might otherwise have resulted from exposure to a carcinogen. That is, a carcinogen can cause a cell to become, in essence, to be malignant. But that cell is attacked by lymphocytes and removed, and killed and removed from the body before it can develop into an overt cancer, before it can multiply and continue thereon. That process has nothing to do

A. (cont'd.) with the amount of a carcinogenic agent in the body. It has to do with the process of cancer itself.

Q. There are, though, defence mechanisms in the body which act against asbestos?

A. Right. There are enzymes that are involved in DNA repair. There's a lot of processes in the body that are protective and will certainly reduce the risk of developing cancer where they otherwise could be absent.

Q. It's possible that some of those defence mechanisms may be very, very effective when you are talking about very low doses of asbestos?

A. The possibility of some of them being dose-related exists. I don't know of such a dose-related process for asbestos, and I don't know if anyone else does either. I would be interested if you could provide animal data that would show such a process to operate.

In some cases, with chemical carcinogens, processes have been proposed that operate better at low levels than at high levels. That is, you can metabolize through certain pathways carcinogenic agents that are, that saturate and thus at higher levels are less effective.

But I do not know of such processes for asbestos.

Q. Yesterday you mentioned a study that the Selikoff group had done of neighborhood residents in Patterson, New Jersey, where people who lived near a plant in the Riverside neighborhood, I believe it is, were compared with a control group in the Totowa neighborhood, is it, farther from the plant?

A. Yes, Totowa.

Q. I believe that study, I believe first of all, is published in the New York Annals, volume 330, the Annals of the New York Academy of Science, Volume 330, page 417. I

Q. (cont'd.) think it may have been cited yesterday. That study looked...I believe the study, you just mentioned it briefly yesterday and maybe I should give you the chance to explain it.

A. I was asked about it. I was asked if I were aware of any neighborhood study, and that was the one that I was aware of.

Q. I believe that study was unable to detect any increase in deaths or deaths due to lung cancer, or deaths due to cancer, in the exposed group living near the plant as compared to the control group living farther from the plant, is that correct?

A. That's correct. The group that lived within half a mile of the plant...I pointed out that the exposed group was largely resident within a quarter of a mile of the plant, and that would encompass less than one-quarter of the total population. The power of that study to detect a significant level was very limited, because the small numbers of deaths involved...that is, one would have to continue it for, one would ideally need much larger populations or a much longer followup to have the ability to detect the very low changes that might be expected from some of these neighborhood exposures. None have been seen at this...

Q. Nonetheless, given the limitations, there was no increased risk found?

A. That's correct.

Q. Is followup on that study a reasonable course to answer the question which I guess most of us are most interested in now, which is, how much risk is there at lower levels of exposure?

A. I think that the study shows that there is not a tremendous risk of lower levels of exposure, and that's what the followup would eventually show. I don't have the study.

A. (cont'd.) What page is that?

Q. Page 417.

A. Well, in this study there were forty-one deaths from lung cancer, which would be the site that one would expect that one would look to to ascertain whether there was an excess of not. If the exposed group comprised one-quarter of that group, even a doubling in the risk would not be seen at the ninety-five percent certainty level with the data that are here, so the power of the study under the conditions, in essence, says there is not a doubling of the risk of lung cancer from that neighborhood exposure. And that's about all you could say from that study.

It does not have the power to provide information at low risks that are obviously the issue of concern.

Q. You refer to one-quarter of the people in the Riverside area as being the exposed group. It's not discussed in the article that way, is it?

A. That is not in the article.

Q. The article talks about everyone in Riverside living within a half-mile of the factory and being referred to as the target area?

A. That's the group that was followed, but when you look at the exposure pattern, the only area that you could identify excess exposure in, on the basis of settled dust, was in the group that were within one-quarter of a mile of that factory. There can be some, but extremely minimal exposure, that was indistinguishable from the background with the data that were available for those resident one-quarter to one-half mile.

Q. You are talking of that...

A. And that's...just exists in the common sense that there is certainly a gradient as you leave a source.

Q. You are talking about settled dust

Q. (cont'd.) measured recently?

A. Yes. We measured to assess the exposure of these residents. We measured the fraction of amosite asbestos in settled dust in the homes. It wasn't a measure of the air in the homes, it was simply a measure of how much relative exposure different homes had at different places.

Q. But that is not to say that there may not have been much higher concentrations throughout the whole Riverside area during...

A. Yes, it is, because we made...

Q. ...earlier years?

A. We made the comparison with homes outside the Riverside area. That was the point. We measured the settled dust in Totowa, we measured the settled dust in different distances from the plant in Patterson, and we measured the settled dust in homes of workers.

In the group that lived within a quarter of a mile, the levels rose considerably to about four times that which was measured in the more distant group, that is the group within a quarter to one-half a mile, which in turn were not distinguishable from the amount of settled dust per unit collected volume in the Totowa or other distant areas where comparisons were made.

Q. These are measurements made twenty or thirty years after...

A. No, they are measurements made in the last five years.

Q. Which was twenty or thirty years after the plant was operating in this area?

A. Yes, that's correct.

Q. The relevant period for exposure...

A. But it's the only measure we have. We are not there forty years ago to measure the air levels.

Q. I gather that this more recent measurement is, as I have indicated, not in this ...

A. This is a study of the mortality experience, and that's what that paper is on.

Q. In this paper they don't make a distinction among the different residents of Riverside?

A. They do not.

Q. Just out of curiosity, this paper, as with many papers you discussed yesterday, were published in the Annals of New York Academy of Sciences. Is that correct?

A. Yes.

Q. That's not a peer review journal, is it?

A. It's not in the sense that there is...each article is sent out individually to a group of people representing a given field. It is reviewed by a committee of the New York Academy of Sciences that both, at the time that the conference is established and subsequent to that. That Annals was the proceedings of a conference held at the Academy in June or July of 1978, an advisory committee selected the participants and reviewed the program. Following that review, the participants, during the conference, presented the papers and they were submitted for publication.

There was considerable peer review at the time of the conference itself, with participants criticizing or otherwise discussing the papers extensively.

Q. Those discussions, are they published in the Annals, too?

A. They sometimes are. I don't think they were with that one.

Q. I would like to talk for a little while about exposure information in epidemiology studies. Perhaps we could start by looking at the way the exposure information was collected in Corbett McDonald's study of Canadian miners. Are

Q. (cont'd.) you aware of how he collected his exposure information?

A. A lot of dust counts were taken of the total particle concentration in different mines and mills over the years by Lachance, and largely Maurice Lachance, I guess... I think his name is. They utilized that to establish a dust index. They attempted to associate jobs with his exposure measurements.

Q. As I understand it, and tell me if this is your understanding, in addition to identifying job types and identifying exposure levels for each job type based on thousands of dust counts, in the McDonald study they calculated for each man in the cohort the amount of dust he would have been exposed to, given his job history from job to job over the years, and given the information they had on how much the dust counts were for each job for each year.

A. I think that's correct. They made an estimate for different...I think probably, if I recall, it was on an individual basis and they categorized those individuals into groups according to those estimates. There were no measurements made for an individual. They were simply estimates based upon the dust counts taken in similar jobs at different periods of time.

Q. As I gather, as they divided the types of jobs into I think more than five thousand types of jobs, they attempted to determine as precisely as they could what the dust level would have been for each man during his working career?

A. Presumably that was the case. That's what I believe they stated.

Q. I believe you used a different method to determine exposures in your study of Canadian mines, is that correct?

A. We used two methods.

Q. Perhaps you could explain how you calculated the exposure value.

A. In our case we had made measurements in the mines in different work activities, with fiber counts. These were recent measurements and thus the estimates that would obtain for earlier years were largely based upon Gibbs' descriptions of the changes that took place, and his changes in dust counts. Thus we used an average in the estimates that I made in this particular document, used the average exposures that we had measured in the 1974 period, 1973/74 period of time, and estimated that they could have been approximately three times as great in earlier years, which is a reasonable estimate for the change in the average according to the trends.

The concept of dealing with an average of a group rather than attempting to do it for each individual in the group differed from McDonald. We also did it his way, but in both ways...and indeed in McDonald's himself, these are simply guesses as to what the persons and the group were exposed to in earlier years.

Q. But their guess...in McDonald's they attempted to get as much data as they could about exposures on a job-for-job basis and on a person-for-person basis?

A. They couldn't do it on a person-for-person basis. They could do it on a job-for-job basis. We had the same data and we looked at the same data.

Q. They knew which persons were in which jobs?

A. We did too.

Q. In the McDonald study.

A. We did too. We had a complete work history of virtually everybody in the cohort that we studied, except for the few people that numbered less than one hundred, that were

A. (cont'd.) deceased at the time, and we had information on those from the union records as to what was the work history as they knew it. But there could have been some error there.

Q. Have you recorded the data from that...

A. No, because it really doesn't have that much...it really doesn't make that much difference. In fact, those data were used in the dust estimates that I characterized people in the presentations before the Beaudry Commission. The estimates from the averages and the estimates on the individual jobs that were made, we had more jobs lined up than you could shake a stick at, and it really is an exercise in numerology that doesn't have that much bearing in reality.

Q. Do you think an average figure for a cohort tells you as much as a detailed, painstaking attempt...

A. It probably does.

Q. ...to recreate...

A. In general, for the limited information that we have...particularly when the information is based upon dust counts that have no bearing upon the biological effects...that is, the total dust, for example, in a drilling operation is quite different in composition from the total dust cloud in a bagging operation. At the time that McDonald made these estimates, they were based...and still are...based upon the total dust rather than upon the fiber count. The fiber percentage in each of these different work activities varies considerably, and he even indicated that it was not possible to make meaningful comparisons on a job-by-job basis, to fiber counts. That created great limitations in this, what would appear to be very accurate, process of assigning a dust exposure. In fact, it isn't that accurate and I think McDonald would recognize that it isn't that accurate. It was the best he could do.

DR. UFFEN: He did.

THE WITNESS: Pardon?

DR. UFFEN: He did.

THE WITNESS: Oh.

MR. HARDY: Q. Let me ask one question though. I believe Dr. McDonald has stated in his writings and stated again to this Commission last week, that one of the reasons he would have some confidence in the dose and exposure information he collected is that he has analyzed it a number of ways, and the dose-response curve always appears to be fairly consistently there and fairly consistent from analysis method to analysis method.

Now, from your data you can't check...from the way you have constructed the average exposure for the mines, you can't do those sort of cross-checks to determine whether your exposure information is correct?

THE WITNESS: A. Yeah, we found those that we estimated to have higher exposure, to have higher disease. In the highest-exposed group, we had a fifty percent excess mortality compared to the lower-exposed group. There was a significant dose-response relationship according to the estimated individual dust exposures.

Q. You mentioned as we were discussing your study, the presentation you made to the Beaudry Commission several years ago. I believe you discussed in that presentation your Canadian miners study, is that correct?

A. Mmm-hmm.

Q. In your current tab nine document, do you use an average exposure level in 1973 and 1974, in the mines, of fifteen fibers? Is that right?

A. That's right. That was the average of the measurements that we made, weighted according to the number of people that would be working in the area at that time. That is the...

Q. Do you remember what average figure you used

Q. (cnt'd.) when you appeared before the Beaudry Commission?

A. No, I do not.

Q. I believe on page forty-four of that presentation you used the figure twenty fibers per millilitre as the exposure in the 1973-74 period of time?

A. Is that an average of the counts or an average according to whether people were working in that area or not?

Q. I can read you what you said, and I'll let you interpret it.

A. All right.

Q. "Exposure in the 1973-1974 period of time is such that twenty fibers per millilitre would not be an uncommon air concentration in mines and mills."

A. Okay. That's absolutely true.

Q. You go on to say, "Historical reports on dust concentrations indicate that these concentrations are much lower than those that have existed in the past, and extrapolations from dust data would have some job areas with in excess of one hundred fibers per millilitre, probably the exposure of past years, in the 1920's and 1930's, for example."

A. Yeah, that's correct. That twenty fibers is not an average. Many jobs were measured with that level or higher, and some were even...one count we had was even in excess of one hundred. There is nothing that is incorrect about that statement.

Q. Perhaps you could explain to me where you get the fifteen? It's an average of what?

A. It's the approximate average of dust counts

A. (cont'd.) that we took in the mines and mills.

Q. Does that average take into account that more men are in some areas?

A. Yes, it does. Let me make sure, how far to reconstruct it, even.

MISS JOLLEY: What page is this on? I'm sorry.

MR. HARDY: The Beaudry information?

MISS JOLLEY: Oh, no. Fifteen...no, the fifteen fibers.

MR. HARDY: Page thirty, I think.

MISS JOLLEY: Thirty?

THE WITNESS: A. I averaged the data in the one mine that contributed roughly ninety percent of the workers in the cohort that we studied. The addition of the other data would provide little change whatsoever from the...

MR. HARDY: Q. Are you talking, perhaps, about table three in tab eight?

THE WITNESS: A. Right, table three in tab eight. The average for mine two was fourteen point zero. The addition of the...weighted by the persons for the other mines...would change little that value, and thus I used an approximate value of fifteen.

Q. You say the average was fourteen?

A. Of those samples that we took, those samples were taken largely to reflect the number of people that were employed at a given period of time. So it is in that sense largely a person-weighted average, but a very subjective one. We did not have access to the entire work force over the entire working day so that we could sample everyone. But in that period of time I feel confident that roughly fifteen fibers would accurately reflect the average exposure of people working in the mines at that time.

Q. Wouldn't, obviously, reflect whether there

Q. (cont'd.) were some workers who had much higher averages and other workers who had much lower averages?

A. Yes, and that's reflected in those data. I give the mean and range, and some people certainly had higher exposures and some people certainly had lower exposure, and that's evident.

Q. But that sort of dose distinction among workers was not used by you in then constructing a dose-response curve from this data?

A. I didn't construct a dose-response curve from this data.

Q. In this article, tab eight? That's right, there's no dose-response information in this tab eight, is that correct?

A. That's correct.

Q. But in tab nine, you do construct a dose-response curve from this data?

A. I did not construct a dose-response curve in tab nine either.

Q. Well, in tab nine don't you...when we get back to table fourteen...predict how much risk would be created by one fiber per millilitre per year?

A. Yes, but that was not done with the dose-response curve. I used the average exposure of the group as indicated there, and the mortality experience of the entire group.

When you are dealing with things that are linear, and the linearity was established as a premise earlier on, then...

Q. In other studies?

A. In other studies, including McDonald.

Thus, when you have linearity, one can utilize averages for dose-response information. Because..

Q. Assuming there is linearity in all of the

Q. (cont'd.) various cohorts?

A. Yes. For those cohorts for which we do have dose-response information over a range of exposures, that was the case...within the variability of the data.

Q. Given that outlook on predicting risk from epidemiology studies, would not it be possible to go back and collect all of the epidemiology studies that have been done of asbestos, many of which are not covered in your review, to make our best guess of what the average exposure of the cohort was?

A. If there were...

Q. And similarly calculate these numbers as you have calculated them?

A. Right. If there were some information on dose, I would have done that.

Q. There is no information on dose on any case?

A. That I had access to. There was not studies that I had access to that would have been of significance for this review.

Q. You didn't look at any studies other than the ones that are included...

A. Yes, I did. I looked at Mancuso's studies, I looked at the update of that where there was considerable disease found, including mesothelioma, in a chrysotile friction products plant, but I could not establish any dose information there.

Q. Did you consider at all going back to the investigators to further the scientific inquiry to find out as much as we could...

A. I was unable to do that in the time constraints that I had.

Q. One study you don't talk about in here, that I'm sure you've seen, is the recent study by Newhouse and Berry,

Q. (cont'd.) of the friction products plant in the United Kingdom. Was there some particular reason that wasn't included in this review?

A. Which study is that?

Q. It was presented in Cardiff, at the conference last fall by Newhouse...

A. Oh, I was not at Cardiff, and I'm unaware of it.

Q. I see. I won't ask you any questions about that. The Commission has heard about it and I was just curious why it wasn't included there.

A. Thank you.

Q. Let me ask a few questions about the exposure information you used to predict risk from Dr. Selikoff's insulation worker study. I gather there also we do not have individualized exposure data based on the workers life histories or measurements made in the areas where those workers worked over the years?

A. We have only measurements in the typical job activities they were engaged in. In that study, as in other studies, there was no information whatsoever on any individual's past exposure history.

Q. In fact, we have no measurements of areas where those particular insulation workers were in any particular period of time?

A. In some of them we did. We were measuring exposures in the 1968/1969 period of time, in individuals that happened to be in his cohort. But there was no identification made of, the individual per se was not...the measurements on an individual per se were not utilized for that individual. That would be totally nonproductive. We would be dealing with a scant handful of people.

Q. So I gather that with that study, as with your mining study in Canada, what you have done to predict risk is picked an average figure for the entire cohort?

A. Yes.

Q. Given that average, you cannot really tell for sure what the real actual, were it to be known, dose-response relationship is among workers in that cohort?

A. No. We have no data on the effects of differing exposures.

Q. One other study you use in your review in tab nine, is the amosite factory study. I believe it's accurate that there is no exposure information on that plant directly, is that right?

A. Correct.

Q. I gather what you did was went out and looked for another plant?

A. There were two plants that had been studied by NIOSH, the data from which are published in that document that you were reading from earlier - the 1971 criteria document of NIOSH.

Q. When were those plants studied?

A. In 1968 through 1970.

Q. When were the relevant exposures in this amosite factory plant?

A. In 1945 through 1954.

Q. As I read your document, your assumption is that the exposures measured in the late sixties in two other plants were probably very similar to what occurred in the 1940's in this New Jersey amosite plant?

A. Yes, I believe that to be the case.

Q. Maybe we could just look at the data from one of those plants, for a minute. I believe it's in tab nine, and it's table ...

DR. UFFEN: Table five.

MR. HARDY: Table five, right.

MR. HARDY: Q. I believe this is the NIOSH data, actually from the 1972 criteria document we were discussing earlier, on two plants, both insulation plants, monitored in 1967, 1970 and 1971. Was this amosite insulation that was being monitored in these years?

THE WITNESS: A. Yes, it was.

Q. Looking at the top table, plant one, isn't it true that there was a substantial reduction in exposure levels between 1967 and 1970, at the Port Allegany plant?

A. There was a reduction in 1970 there, which again rose in 1971. Some of this could be the result of just different averages. Part of it was the result of an attempt to install dust controls sometime during that period of time, which proved to be relatively ineffective and led to the closure of the plant because it was not able to control the dust.

In fact...

Q. I notice that, for instance, mixing was measured to have a mean of a hundred and seven fibers in 1967, and only twenty-seven point seven fibers in 1970?

A. Right. There were two counts made in 1970, and there were three counts made in 1967.

Q. Similarly, forming drops by a factor of four between 1967 and 1970, from ninety-eight point nine fibers, to twenty-four point one fibers?

A. Correct.

Q. Given these sorts of variations over a short period of time in this plant, different from the plant in New Jersey where we have the medical evidence, isn't it very questionable to be able to estimate from this information what might have been the exposure levels thirty-five years

Q. (cont'd.) earlier in an entirely different plant?

A. I think it's the best that we can do. We cannot be absolutely certain that it's correct, but it provides an estimate that is likely to be fairly accurate in terms...and here I mean within a factor of two, of what the average exposure would be. In no case are any of these estimates likely to be more accurate than that, nor are any of the estimates in any other published documents likely to be more accurate than that.

Q. Are you saying that the sort of estimation of exposures that was done for this amosite factory plant are as good, based as they are on exposures in different plants thirty years late, not calculated on an individual basis, as the type of exposure information collected through careful discussions of individuals and many monitoring points in the Canadian miners study of Dr. McDonald?

A. I think they are better in terms of fiber count.

Q. Then I gather you would believe that Dr. McDonald spent a lot of time collecting a lot of data that isn't very useful?

A. He spent, I think, more time than was necessary for the accuracy of the data that he obtained, because he obtained data in terms of particle counts. Its conversion to fiber counts is not possible in any accurate way for each of those jobs without enormously great effort in reconstructing situations in previous years. Since it's the fiber counts that are important, that information that he presents is of limited relevance. It is the best data that exists, and I'm not belittling what he has done. But it is not something that has a great deal of accuracy associated with it.

Just because a considerable effort went into collecting dust counts doesn't ascribe to them some magic

A. (cont'd.) of accuracy in terms of fiber counts.

Q. I noticed in tab nine you do make that criticism of Dr. McDonald's study and also Dr. Enterline's study. You don't make that criticism with respect to Dr. Dement's study, but it, too, was based primarily on particle counts, wasn't it?

A. Yes, it was. But there were fiber counts that were made over a much...parallel counts, a much greater number of parallel counts made there. To a limited extent, it also obtains for that study, but to a lesser extent because there was a greater number of measurements. He had more than one thousand that he reported as being used to compare fiber and dust counts, and it is that comparison that is important.

Additionally, that plant had relatively few processes. It didn't have the five thousand different job activities and twenty different mines, or however many different mines there were, that McDonald had to deal with. McDonald had to deal with an enormously complicated and complex situation, whereas Dement had to deal only with a fairly simple situation.

DR. UFFEN: Counsel, could I sneak a question in that is related?

MR. HARDY: Certainly, Dr. Uffen.

DR. UFFEN: Perhaps others are getting a little confused about fiber counts, particle counts and weight or mass counts, nanograms.

Could you tell me at this stage, if the fiber counts are the ones that are important for the health effects, why you people still make nanogram measurements?

Or to put it another way, what's the best way in the future?

THE WITNESS: Yes. I think...I have not made that much of an effort to investigate possible alternatives to phase contrast fiber counting in the workplace at this time, so I don't want to make a statement that would say that that is the method that undoubtedly will be used for the next ten years

THE WITNESS: (cont'd.) or so. There may be something that some people have worked on that's really great that I'm just not aware of.

But at this point that appears to be the way one will go in assessing the workplace, because it's simple, you can...it bears upon previous data that we have. What we are looking for is methods to achieve a reduction in workplace air concentrations, and as variable as it is and with all the problems that it has, I'm unaware of a better method for rapid and economical assessment of the workplace. That is a method that only counts those fibers that are longer than five microns, it does not make an attempt to identify them as an asbestos fiber per se. That assumption is made on the basis of the process. If you were going into an asbestos factory, the chances are that virtually all the fibers that you would be counting would indeed be asbestos, and thus unless there is very complicated work situations there is not a need to discriminate between fibers of one type or another. Even there, perhaps in health effects that would even be unwise.

So that's a method we have at this moment for the work place, and it certainly is a better method for assessment than the old method of particle counting which assessed an environment in terms of the total dust concentration present, and which in terms of asbestos health effects could be inaccurate because, as mentioned earlier, there is a lot of host rock dust in one case in the drilling and mining operations, compared to much less so in the milling, and particularly the end process of a mill. When you go to manufacturing a lot of other dusts - talc, silica, calcium silicates - are used that are also counted in that method rather than simply the fibers, and their biological effect is certainly less than that of asbestos.

Thus, the fiber counting is a significant step forward from that.

THE WITNESS: (cont'd.) Okay, now let me just go to...I wanted to discuss that before, now I'm discussing environmental measurement.

There one is faced with considerable problems because you cannot use phase contrast microscopy for two reasons. Firstly, when you are making an assessment...firstly, the volume of air that one would have to collect to provide a countable number of fibers is considerable. In principle you can do this with large pumps, and simply count, but that's one minor limitation.

The more important limitation is that most of the fibers that you count at the levels that you are measuring asbestos in the air are organic fibers and fibers of other types. Without a great deal of trouble, using distortion staining and petrographic microscopic techniques, you cannot determine with phase contrast microscopy whether those are asbestos or not, and thus a simple counting of fibers in environmental circumstances has no relationship to the amount of asbestos that's there or the number of fibers of asbestos that's there...

DR. UFFEN: A very poor relationship.

THE WITNESS: At the level of less than, say, point zero three fibers per millilitre, in the outside air situation.

Additionally...so therefore electron microscopic techniques have been utilized, and these can be of two types. One there simply counts all fibers and after processing a sample to allow their being viewed, this has two problems. It most heavily weights the very small fibers. That is, you've got a little fibers and if you count every fiber, you can get enormous fiber counts based upon the presence of these minute angstrom long...tenth of a micron long fibrils.

Additionally, chrysotile is a very friable fiber, and in order to identify the fibers in air samples collected in the ambient air you have to disperse the sample because there

THE WITNESS: (cont'd.) is so much other garbage present to which the fibers adhere, that obscures their viewing in any direct transfer process.

In the dispersal of that material, either with ashing or ultrasonic techniques, you break apart the fibers, and so counting the fibers can in fact overestimate their number by virtue of that number being increased through the processing.

So a fiber count of the ambient air level, even if you could make a conversion between electron microscopic fiber counts and optical fiber counts, would be a difficult and misleading...could provide misleading information. It would be highly variable.

Thus we adopted the method of quantitating all asbestos by mass, and that's the data that I presented here, the data of the other groups that have been measuring asbestos in the ambient air - Battelle, for example, also utilizes a mass measurement.

This is, I believe, a more representative measure of the health effects in that it attributes greater importance to the larger fibers directly proportional to their mass, and does not give a high weight to these little tiny fibrils that are ubiquitous...

DR. UFFEN: You still have to have the electron microscope...

THE WITNESS: Yes.

DR. UFFEN: ...and that's the slow, cumbersome, expensive part of it.

THE WITNESS: Right.

DR. UFFEN: You still have to sort out how much other kind of silicate might be there along with asbestos.

THE WITNESS: That's correct, and that's where the problem comes.

DR. UFFEN: Would it be fair enough for a person

DR. UFFEN: (cont'd.) like me to come to the conclusion that for the foreseeable future we had better use both fiber and weigh them?

THE WITNESS: For environmental measurements when you are making an assessment of the possible impacts of a given situation through measurements, and here I certainly advocate the need for air measurement.

For example, air measurements in schools to get a feeling of what are typical air concentrations found in those circumstances where one sees damage...to obtain an assessment of that or buildings or whatever.

If you wish to find out what are typical average concentrations in schools, a program where sampling is done to be representative of the situation is an appropriate one. In other words, to have a program that will allow a problem assessment as opposed to an individual school assessment. That's the distinction I'm making.

There, measurements made by the electron microscope are the only ones that I think are valid. We did do a study...in fact it's in here...where we compared the fiber counts in buildings where we had corresponding electron microscopic measurements of the total mass, and there was no relationship whatsoever. They were all over the lot.

DR. UFFEN: Thanks, counsel.

MR. HARDY: Certainly.

DR. UFFEN: You can have fifteen minutes of our happy hour.

MR. HARDY: I doubt whether I'll even need it, but thank you, Dr. Uffen.

MR. HARDY: Q. Are you familiar, Dr. Nicholson, with the Simpson report? The Report of the British Advisory Committee on Asbestos?

THE WITNESS: A. I'm familiar with it. If you

A. (cont'd.) are going to ask me details on it, I'm afraid that I'm a little...

Q. I don't think any details. It's a very basic question.

As I understand it, that's a document that was prepared over a couple of years by a group of, a broad-based group, including a number of medical experts, giving advice to the British government on various issues relating to asbestos?

A. Yes.

Q. As I read the report, there were a number of medical experts who worked on the report as part of the Committee and as part of a drafting group and as advisers?

A. Yes.

Q. Do you recall that in this document there is a risk extrapolation which attempts to predict the risk of asbestos at current occupational levels?

A. I think...if I recall...I think Peto's estimates might be in there in terms of risk extrapolation. They also discuss Enterline's and McDonald's studies on dose-response.

Q. That's right. When this Committee in the United Kingdom decided to discuss dose-response and predict low level risks, they looked at three studies, and those were three of the studies that you find least appropriate to look at, in your tab nine?

A. Yes, for the reasons I gave.

Q. So you would disagree with this group on what studies should be looked to to make those sorts of decisions?

A. No, I think one should look at all of the information that is available. Some of the information that I have included was not available to them, and to that extent I would disagree with conclusions that they draw based upon those three studies, because there are problems with those three studies, particularly there are problems with other studies

A. (cont'd.) as well that I think are...well, there are problems with all studies. To the extent that we are trying to see that what is the circumstance in as many...the situation in as many circumstances as possible, it is most appropriate to look at as many circumstances as possible.

Q. They didn't discuss, in terms of dose-response relationship or risk extrapolation, Dr. Selikoff's insulation workers study though, did they...in the Simpson report? And it was certainly available to them at the time.

A. The...I guess the two studies they utilized were those that had published dose-response curves, and they might have selected them for that reason. I don't know what their selection criteria was. One might ask them about that.

The other study, that of the few people that were at work in the Rochdale plant, were utilized obviously because it was a British study available to them.

Q. With some dose-response information, I believe?

A. Yeah, with...not so much in the sense that you have been using it, they didn't have dose-response relationships. There they only had...

Q. There they had individualized workers...

A. They only had averages, and they used averages.

Q. But they had averages measured over a number of years and took into account those changes in exposure over the years in calculating total dose, isn't that correct?

A. Yes.

Q. But I gather you would disagree with the proposition that the most appropriate data for doing risk extrapolation would be that data which includes information on dose-response for workers within the study?

A. Say that again?

Q. I'll try to make it clearer. You would not agree that risk extrapolation most appropriately should be done, when it is available, from studies with dose-response information within the cohort?

A. Oh, I think you would want to use that as much as possible. That would be the most desirable information.

But if that information has other difficulties associated with it in terms of what that dose-response parameter is, or the dose parameter is, you have to recognize that problem and not ignore it and not put your head in the sand and say because that's what's published, that's the best information, that's the only information I'm going to use.

Q. You make an additional criticism of use of the McDonald data in your tab nine, and maybe I should point out the page just so it's clear to everybody. I believe it's on page twenty-one of tab nine.

You indicate that the relative risk of lung cancer for various exposures, in the McDonald study, were obtained through a case-control procedure using all cohort members, and not by comparison to an unexposed population.

Do I gather correctly that within context that's a criticism of the data, the analysis by Dr. McDonald?

A. Well, he's making comparisons with others that could have exposure as well. In particular it's an internal exposure control.

Q. That's correct.

A. As opposed to looking at what would be expected in a population in the absence of any exposure whatsoever.

Q. Well, I have a couple of questions about that.

First of all, isn't it true that he analyzed his cohort both ways? Both in terms of comparison to the general population...

A. Of Quebec.

Q. And in terms of a case-control study within the cohort?

A. Yes, that's true.

Q. Therefore, the fact that he did this additional analytic step isn't really a valid criticism since he also did what you would like to have him do?

A. He did it in comparison with the population of Quebec, which in an early study he acknowledged would have at least fifty percent more cancer, and lung cancer in particular, mortality than the counties in which the mining populations lived.

Q. I'm not asking that. The question I...I am trying to understand why you are crticizing his use of case-control study?

A. Because the internal control may give an underestimate of the risk. The external control which he used, and which I didn't criticize and I didn't use..that is, I didn't even want to get into the criticism...of the gross underestimate that that provides because of the fact that the data that he used for that external control were not relevant to the population, would appear to be not relevant to the population that he had under study and which he, himself, had stated to be the case in his first publication in mortality.

Now he did that because I think that's the only data that were available, but we couldn't get the data of the local counties in order to make that external comparison ourselves, so I'm not saying that he did something wrong. I'm just saying in what he did, there are limitations. And that's the problem with any epidemiological study. There are limitations no matter what you do.

Q. My question is to try and understand your criticism of his case-control.

A. I explained it.

Q. Well, I think you told me about problems with the general population control?

A. No, you brought up the general population. I was only speaking about problems with that particular method.

Q. Why don't you say it again, since I missed it, what's wrong with his having done an analysis by use of the case-control study within the cohort?

A. Because one is not sure that, particularly in that group that was working in those mines in that town, that there was no exposure even at the lowest exposure group.

Q. Oh, I think...let's go back...

A. Have you ever been to Thetford Mines?

Q. Let's go back a step. Do you understand why he did the case-control within the cohort?

A. Yes, I do.

Q. What's your understanding of the reason he did it?

A. To the extent possible, that that provides information on the effect of added exposure.

Q. In other words, he was attempting to see whether he found the same dose-response curve by that method of analysis that he found by the other method of analysis he used, is that correct?

A. Well, it provides additional substantive data on dose-response effects.

Q. As I understand it, there's some valuable aspects of this sort of analysis, meaning, for instance, that by ...well, let's start this way. What he is comparing in that study is the exposures which he knows the lung cancer victims had, to exposures among other people within the cohort who don't have lung cancer. Is that right?

A. That's right.

Q. And trying to see whether there are higher exposures in the lung cancer people which would, if you correlate...

A. He looked at the relative risk of people characterized by different exposures, in lung cancer victims and non-lung cancer.

Q. One of the advantages of doing the case-control method within the cohort is that various normal problems with retrospective cohort studies, like health-worker effect, or confounding variables, or unusual lung cancer rates in particular communities, in theory are controlled for?

A. It does do that. That's right.

Q. So there is a valuable reason for doing that?

A. I'm not denying that.

Q. Well, I guess you are not denying it now and maybe I overread what you stated on page twenty-one of tab nine, but I read that to be a very critical comment on what Dr. McDonald had done.

A. I didn't think it was that critical. It just acknowledges that even within that group there can be some effects of exposures that are unaccounted for.

I used the data because it was the best data that were available, from his study.

Q. Let me ask you about a concept which you discuss in tab nine, and that's the concept of saturation. First of all, I guess, is that your term?

A. I'm sure it's been used previously. It's not a very clear term. I mean, the term is not very clear. The phenomenon is...what is happening is also not clear.

What one sees is the...in cohorts, you see it to some extent in McDonald's group with the highest exposure. You certainly see it in Seidman's, you see it in some other

A. (cont'd.) exposed circumstances. It, in part, may be the age effect which is much more universally seen, that I described yesterday, where after the age of sixty there is, for whatever reason, a significant decrease in the relative risk of people exposed in the same circumstances as people who died earlier. As these cohorts were described by their cumulative dust exposure, that would have the oldest people being in the highest dust exposure, and that effect might be part of the reason one sees a lessened risk there.

The fact is, and that's all I was simply saying, is there appears to be in many of the studies a reduced risk at higher levels, relative to that which one sees at lower levels. Since we are concerned with attempting to assess effects at lower levels, it's important that one recognize the possible reduction at the high-dose points, and utilize that information that would appear not to be affected by this process that we don't really quite understand.

Q. So you have come up with this term which you call saturation, and I gather it's your term?

A. Not necessarily. It's been used by others. It's a standard way to describe something that flattens off. You talk about saturation of metabolic processes. Other people have used that term in similar contexts. It's not an uncommon term.

Q. As I understand it also, it's contrary to the typical discussion which ...let's start again...it's contrary to the typical concept of a dose-response relationship, that as the dose goes up the response also goes up?

A. It's contrary to a linear dose-respose relationship that would continue forever. You clearly have to have saturation because if you had a linear dose-response relationship, you would soon come to the fact that everybody would die of an asbestos disease. That cannot happen. There are going

A. (cont'd.) to be competing causes of death that will certainly cause a bending over of a curve of asbestos mortality at higher doses.

I mean just that phenomenon alone gives you a flattening of the data in the way that I was presenting it, which was percentages of mortality rather than mortality risks.

Q. Now, I gather that your table six in tab nine is one of the pieces of data that you indicate in this document shows a saturation effect, is that right?

A. We found no change in lung cancer. We found a very flat dose-response curve for lung cancer in that particular population.

Q. Let me perhaps suggest an alternative theory for why you didn't find a dose-response relationship for lung cancer, and that is the possibility that there is misclassification of exposure...?

A. No, I don't think that is the case, because when one looks at the mortality from asbestosis in this group, one finds a very dramatic increase in deaths from asbestosis according to estimated dose. It rises from four percent to forty percent in each...it goes four percent, thirteen...I'll read it, whatever it does...I think it's four percent, thirteen percent and twenty percent and forty percent.

Q. That doesn't necessarily mean that there still wasn't misclassification?

A. The possibility that a single individual might have been misclassified exists. In fact, individuals in a given job...that is, the maintenance personnel which I think were classified in either two or three, depending on their circumstance, whatever they were...could be some individuals in a maintenance graph might spend all this time in the shop, where others are always sent out to deal with something dusty. So that within any category, unless you have personal monitoring from each

A. (cont'd.) individual, you are going to have highly variable exposures. That's the case in any study.

Q. But again, I think one of the things that Dr. McDonald said gave him great confidence in his exposure information was that he was able to find consistent dose-response relationships because of his, I think, the basic epidemiologic principle that dose-response relationships add a great deal of credence and support to associations found in studies, whereas you probably have a different pattern...

A. In the very lowest exposure I'm having as much cancer as he was attributing to the group that he was investigating. And at that point, if you had it continue to rise up like that, the total group would have died of lung cancer that were in the higher exposure category. That is, in the lowest group roughly fifteen percent of the deaths were from lung cancer, which is the same as the highest group that he ever had.

Q. So what you are saying..

A. We are looking at entirely different effects, and the origin of those differences of those effects is not understood. That is, there is no question that the dose-response relationship that McDonald would get, where he would attribute very little effect to a fiber exposure in a mine and mill, is different from any other study that obtains in any other circumstance.

Here we are finding effects manifest at the lowest exposure area, that are comparable to those that McDonald is seeing at the highest exposure category.

Q. And thus he...

A. Unfortunately, we didn't have enough individuals with even lower exposure categories here that would have allowed the extrapolation to a lower risk, or would allow the measurement of the effect of a lower exposure.

Q. So that if there is...I gather what you are saying is that there is some sort of saturation effect that's in effect that would not even apply for the most heavily exposed of the workers in the McDonald cohort?

A. It only began to appear there.

Q. His exposures were many, many times greater than anything in the mines of today?

A. As described in terms of the conversions that he would give, that is correct. Thus the disease per unit fiber exposure that one calculates from his data is much less than one calculates from...and also the data that we calculate in the mines and mills is much less than is calculated from any other study of asbestos products, asbestos workers.

Q. You haven't seen the Newhouse and Berry study, so I can't ask you about that. I recommend you get hold of it, because it, too, has very low risk. The record will speak for itself.

MR. HARDY: I don't have any further questions, Mr. Chairman.

DR. DUPRE: Shall we take a break?

THE WITNESS: Could we make it fifteen, because I have to make a phone call.

DR. DUPRE: Certainly. We'll rise until eleven o'clock.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Mr. Casgrain, proceed, please.

CROSS-EXAMINATION BY MR. CASGRAIN

Q. Dr. Nicholson, in the course of your

Q. (cont'd.) evidence yesterday, you talked about, and I've tried to remember. I don't think I took down exactly what you stated, but you might help me.

In referring to the risk of lung cancer, I think, or was it mesothelioma, I'm not sure...you said it may take up to five to ten years for the relative risk to be manifest?

A. Right. That's lung cancer.

Q. It's lung cancer?

A. Right.

Q. Okay. The question I want to ask you is the following: When you say it may take five to ten years to be manifest, are you saying to be manifest through an examination of the worker, or are you saying in retrospect when you look at your cohort you find that it became manifest at that time?

A. Well, what we are looking at are statistical data on the development of cancer in a large number of individuals over time. What it showed was, what the data showed was, that the relative risk appropriate to a given exposure...and by relative risk I mean the measure of the...what I mean is the observed number of cases seen compared to that expected.

Q. Mmm-hmm.

A. The exact measure of disease potential...and I'm talking about potential, because from a given exposure, the cancers don't appear generally in five to ten years.

Q. That's what I thought.

A. But the probability is manifest such that a certain fraction of them will appear if you have a large enough population, within five to ten years.

Q. Just a second now. I want to make sure I understand.

A. Let me say it a different way.

Q. All right.

A. Let us assume that you have an arbitrarily large population in which there might be a thousand...from a given...and that a given exposure, whatever it might be, if you waited long enough would produce a thousand lung cancers in that population. The distribution of those thousand lung cancers...these are now one thousand excess lung cancers, okay?... this is a hypothetical discussion...is such that some of them would be, would appear in five to ten years, and then would continue to appear thereafter. But of the cancers that are going to come, some will come in that short a period of time.

Q. I see.

A. Now, the course of those cancers would be characterized by the relative risk appropriate to the exposures, be it one, two, three, or be it two, three, four or five, whatever it might be, and the number of cancers will then be that relative risk times what the risk would be of lung cancer in the absence of that exposure...and very few cancers would appear in the five to ten year period of time if the population that we are talking about were exposed at age twenty, because the risk in the absence of exposure was extremely low.

With a thousand people going to die, you might get one or two.

If it were, on the other hand, a much older population, one that were fifty at the time of exposure, the risk that you would be multiplying, with the same amount of asbestos, would still be five, but the underlying risk that it multiplies would be enormous. So you could have a hundred appearing in that five to ten year period of time.

Q. I see. But you are talking about...

A. The number that will appear depends very strongly on what the risk is in the absence of exposure.

But what is important is that we are talking about...if one is talking about causal relationship, because the

A. (cont'd.) data are so scant, I'm fuzzy about five to ten years. But it's very clear that by ten years the biological act...I shouldn't say take place...whatever they might be are such that an exposure can be causally associated in that sort a period of time, and the concept of a lapsed period of thirty or forty years, that has been talked about as a general thing, is simply a reflection of the fact that it may take thirty or forty years for an exposed population with very little risk in the absence of exposure to develop an underlying risk that allows the lung cancer to become manifest. So that thirty to forty years is really a statistical phenomenon appropriate to a population.

Q. As opposed to a clinical one?

A. Yes.

Q. I see.

A. But those that don't get lung cancer in the first five year period of time, there won't necessarily be any clinical manifestation at all, of any change. We are talking in this development of cancer because you don't see anything necessarily prior to a very short period before the... you don't see anything in fact before the manifestation on a clinical basis that would indicate who is going to get cancer years hence. That's an unfortunate thing at this time.

Q. Talking about lung cancer, would you say that there would be a definite relationship between a fibrosis and the cancer itself?

A. Well, I think there is...in those that have fibrosis compared to those who do not, there is a greater probability of developing lung cancer. But lung cancer can develop in the absence of fibrosis.

In fact, in McDonald's survey he had something like...he looked at a cohort, he followed individuals for whom he had x-ray evidence of fibrosis, and found that there was fifty or sixty excess deaths in that group.

A. (cont'd.) Although in the group of all individuals who had died with lung cancer, there were only twenty or thirty...I think it was thirty...that had any evidence of fibrosis. So fibrosis, if you attribute all of those thirty to the asbestos...if you attribute all thirty to the so-called asbestos lung cancers as opposed to the background lung cancers, that is assume that none of the thirty-two that had fibrosis ever would have developed lung cancer, you are still left over with another thirty or forty that developed lung cancer with no x-ray evidence of fibrosis.

Q. But in fact I think you and I can agree that obviously those who have a fibrosis perhaps are more susceptible to being found to have lung cancer in due course, and I don't think you and I are able to establish a real percentage, but certainly...

A. I don't know if you can make percentages, but in all cases we are talking about more exposure, more risk and more exposure is associated with more fibrosis.

Q. Yes. I think in your writings, and I believe also in your evidence, you suggest that the latency period in respect of lung cancer...if we stick to lung cancer...is in the order of between twenty to thirty years? Or is that too wide a gap? Would you say...?

A. That's when most of the cancers are clinically manifest.

Q. That's right.

A. Right. Which is a probabilistic effect. It has to do...

Q. I'm just trying to pinpoint as to when the manifestation occurs. Is it between twenty and twenty-five years? Would you say that?

A. A few can come early, but more come later, and as time progresses the number that are seen increase

A. (cont'd.) considerably. After twenty-five years in a population that was exposed...I'm sorry. After twenty-five years from onset, in a population that was exposed in their twenties...which most working groups, long-term working groups were...is when you see the dramatic rise in the number of lung cancers.

Q. And if you don't see that dramatic rise in a given group, I think it's a good sign?

A. Mmm-hmm.

Q. After twenty-five years.

A. Right.

Q. So that if I was able to come up, for instance, with a cohort of say three hundred and fifty workers who would have been exposed to asbestos, onset twenty years of age, for twenty-five years, and if I found in that cohort not one single anomaly in the x-rays, I could perhaps think it's a good sign, could I not?

A. I would be very surprised if you could have a group of individuals in any of three hundred, in any circumstance, that would not have some anomalous x-rays, no matter where they were.

Q. Is that right? Well, let's perhaps talk about this for awhile. When you say they would have no anomaly, are you saying in effect that most people, in any event, whether their occupation be in asbestos or otherwise, generally have some anomaly on their x-rays, wouldn't you say?

A. The probability of getting some of these anomalies develops with age, and when we've looked at occupational groups...for example, looking at garage workers... one finds that ...and control groups along with them...that both in the exposed and control groups there is opportunity for a lot of asbestos exposures that have taken place in the past, outside of that particular work activity. In the United States

A. (cont'd.) it's largely in shipbuilding. Maybe ten to twenty percent of people...ten percent of people have...of blue collar workers...have had such exposure, and that produces its effects.

Q. I think I misunderstood your earlier answer. I thought I heard you say that you would be very surprised if you found any group with no anomalies, and I thought that you were referring to any group of workers, regardless of their exposure to any kind of occupation. Did I misunderstand you?

A. Well, what I'm in part saying is that you can't...it's very difficult to get a group of three hundred people that have not had exposures of some consequence, or have not had other disease of some consequence that would give rise to other separate, other anomalies on x-rays separate from interstitial fibrosis.

Q. Perhaps I can try to get at it some other way. Taking the ILO classification of the reading of x-rays, which begins at 0 and goes on to to 0 slash one and so on, could we say perhaps that people of, say fifty years of age... my category....if you took their x-rays and applied to them the ILO classification, you would find them with possibily zero slash one?

A. And you would find some with one slash zero or even higher, in any random group of people that you would select in a general population, among whom are people with a variety of possible occupational exposures, or some even unknown.

When we look at the general population of Michigan, I think on those...just recalling, and the data are not necessarily that accurate, but they are not misrepresentative, and that percentage is about right.

I think in those older than forty years of age there was something like five percent...less than ten percent and more than five percent...that had abnormal x-rays categorized

A. (cont'd.) on the IOUC scale.

When we looked at people in New York, the percentage of abnormals has been even higher than if you...even in those that you would not be able to ascribe a previous exposure to. And very older groups, fifty and sixty and up, you can have evidence of fibrosis on x-ray.

Q. Now I understand why you say you would be very surprised if you did...

A. If somebody tells me that they have got three hundred people that are aged fifty or sixty, and there is not a single shred of evidence of any fibrosis in them, I would be very surprised.

Q. Can I change the subject for a minute, and I approach it with some hesitation, because the last time I tried that particular subject I didn't come out too good. Perhaps because of my complete lack of knowledge.

I would like to talk to you about the defence mechanism of the lung.

A. Well, maybe we are both in hot water here. You'll have trouble with it, I'll have trouble with it, too. I'm no expert in defence mechanisms of the lung.

Q. So I guess...well, all right. I was going to try one thing with you. Would you tell me what you make of the defence mechanism represented by the macrophages that I understand have something to do with...do you know anything about that?

A. Yeah, they are a cell that can cleanse the lung of material, and they engulf particles and fibers and remove them via lymphatic drainage systems and other ways, from the lung. And I'm putting it in a very crude way...my description is a crude one.

Q. Sorry?

A. My description is a crude one.

Q. Yes, but is there a size of the fibers that they are better able to absorb, or is there no size?

A. I don't know that much about their ability to deal with fibers of different lengths. They tend to be, I think, in the order of microns in size and the effects of attempting to engulf a long fiber...their ability to remove longer fibers may be reduced because of their own size effect. How much and what the effects of that...what the effects of clearance mechanisms on long fibers is, I don't think there is quantitive data to allow a specific projection to be made.

Q. I asked the question because in the course of your evidence, at least in two occasions, Dr. Nicholson, and I think this morning again, you said something about the small fibers, you know, multiplying in numbers and becoming, as it were, even more dangerous as they became smaller...and you made no reference to the possibility of macrophages being able to take care of some of these small fibers and then through the sputum coming out again.

A. Certainly I think the clearance mechanisms are much greater for smaller fibers. Common sense would seem to have one say that, and that's about all I'm dealing with.

I was also saying that the smaller ones also have a greater potential for penetrability, which I think is again more common sense than...

Q. Than scientific.

A. Than scientific. I am acknowledging that.

What we are talking about are concerns. The greatest concern is, though, that they are there in such great number that even though you are having this clearance mechanism be more effective for them, relative to the larger ones, by the time they finally get to a site of action, there is the possibility of a greater number being there...in fact, that's what one finds. That's what one finds in the pleura, an enhanced proportion of

A. (cont'd.) smaller fibers relative to larger ones, compared to what one finds in the parenchyma. That there are data on.

Thus in the interplay of these different mechanisms, removal which preferentially favors smallers ones, deposition perhaps can go both ways because very small ones can come out and small ones can impact, you have the result that still a considerable number of smaller fibers are reaching cells where potential damage can occur. Their role cannot be dismissed because of that fact, and that's the point I'm trying to make.

Q. All right.

Dr. Nicholson, I believe that one particular segment on which you are an expert is the counting of fibers. Am I wrong? The technology of counting fibers?

A. I was involved in that in earlier years. I mean, I'm fairly familiar with fiber counting, but not so much with new technologies that have been developing in terms of instrumentation other than to be aware of their development in recent years.

Q. For instance, do you know the type of instrument that we now use currently in the mines and mills?

A. No, I do not.

Q. So that you couldn't talk about that particular instrument as to whether...

A. Is that the GCA instrument?

Q. There are...one, I think, was the GCA pumps, yes.

A. Yes. I've seen that instrument and I am familiar with it...for air sampling. I mean, I think that's what you were referring to?

Q. Yes, I was referring to sampling as opposed to counting.

A. Right. Okay. I am familiar with it, but

A. (cont'd.) not to such detail that I could make serious criticisms or apply serious application to it.

Q. When it comes to counting techniques, which actually is really the art of actually looking at the fibers, this you have some knowledge of, though?

A. Some.

Q. Yes? I think perhaps in one of your articles, and I wasn't able to trace it this morning but I'm sure it's part of the tab, you talk about counting and I think you say something about the fact that as you count the fibers, the margin of error increases as the size of the fiber decreases. Is that a correct statement to make?

A. Well, I think there are more errors in enumerating fibers close to the limit of...close to the five micron limit than there are larger. That comes about simply because the smaller fibers tend to have smaller diameters, and when you get to a diameter of the order of half a micron, diffraction precludes your accurately seeing an object.

Q. Am I correct in stating that in effect if you look at the history of fiber counting, by people in general, you may have a margin of error which would be as much as thirty to forty percent to individuals?

A. Oh, sure.

Q. Even more in some cases? One would count, say, three fibers, and one would count zero five fibers?

A. Yeah. And with untrained individuals it could be more. But when you get groups together working on intercomparisons, you can do better than thirty to forty percent, but different laboratories that don't work on the intercomparison program, that would not be unexpected.

Q. Yes, I think it's obvious.

Could I ask another question about your knowledge and mine of matters surrounding this? Are you familiar

Q. (cont'd.) with any of the work that has been done in respect of the chemical observation of asbestos as a result of their finding their way into the manufacturing process, such as cement, for instance?

A. No, I'm not. I don't know anything about chemical alteration of fibers in cement uses.

Q. Can we agree that so far as you know, most of the work is concentrated on surveys, epidemiological surveys rather than biological experiments...at least rather than the chemical change of the asbestos fibers? You have had animal experiments and epidemiology studies, but not much has been done in respect of what happens to fibers chemically after it finds its way into the products.

A. Well, I know of a little research that has been done in looking at the effect of different...some alterations of fiber, but I don't...I mean I don't think there has been that much done in terms of taking stuff that was used in products, separating it and then using that either in in vitro or in vivo experiments that would be focussed upon fiber surface alteration. The possible effects of that...there may be possible effects of that.

I think perhaps the reason that not too much has been done there by the animal people, or the experimentalists, is the finding that implantation studies in other in vivo models, with injection in the peritoneum, for example, that chemistry didn't matter too much in terms of the carcinogenicity of the material. So that's perhaps inhibited the experimentalists in that area.

Q. It is entirely possible, is it not, that the fiber finding itself, for instance, in an acid milieu would perhaps be subject to certain changes and lose, perhaps, some of its toxicity, if I may use the word?

A. It could. Certainly one can find biological

A. (cont'd.) changes in in vitro systems according to surface property alteration. Either ball building, for example, where it's just physical alteration of the surface, or coating the fibers with...

Q. Resin?

A. ...methyl cellulose, for example, is another one I know of, has affected their ability to red cells, so there is a surface phenomenon acting upon a cell membrane, that appears to be related to...

Q. May I turn to table fourteen of tab nine, exhibit nineteen, Dr. Nicholson? That is the table where you take all of the studies and you give us percentages of expected lung cancer deaths, for instance, and then percentages of observed deaths for all asbestos disease, and so on and so on.

When I look, and you know I'm more concerned perhaps than others with the McDonald study, because it did concern the mines in Quebec, and this is why I wanted to ask you about this one in particular. At the bottom of it, I see here that when you deal with asbestos disease and you talk about the percentage of risk per fiber year, your study comes, if I read the figures properly, to something like five times as much in terms of percentage than Dr. McDonald and Liddell's Study.

Perhaps you have explained that already, but I would like you to tell me perhaps why come to that, and before you start I want to tell you something else, too. I want to ask you something else.

When I look at this table fourteen and I see these figures, are those your extrapolations from the figures of the various studies, or are those extrapolations made by the people who made those studies?

A. They are my figures from the data presented by the various individuals, and the method by which I arrived at it was described as I considered that particular study. I think

A. (cont'd.) I presented all the data in here that allowed the estimates that I have in table fourteen to be correct. But it is mine, that's what I'm saying.

Q. Not that I query your ability to calculate, which is obviously much better than mine, I just wanted to make sure that...

A. Well, I've been known to make a mistake. At least I thought I made a mistake once, but I was wrong.

Q. Well, I'm wrong all the time. I just wanted to make sure that the figures I see here when you compare your studies and that of Dr. McDonald, are figures extrapolated by you, not by Dr. McDonald?

A. Correct.

Q. All right. Having said this, I now come back to the first question which is, why is it that you come with five times greater risk than Dr. McDonald in respect of asbestosis?

A. I touched upon that yesterday...

Q. Well, I must have missed it.

A. Let me just say what I said then, and that was, we looked at Thetford Mines and McDonald looked at Thetford combined with Asbestos, and he stated that...and his explanation was not forthcoming...that is, he was unable to explain the differences...but he found a greater mortality in Thetford...

Q. Than in Asbestos, that's correct.

A. ...and greater effects in Thetford compared to Asbestos, whatever was doing it. So that effect might explain part of the difference.

Additionally, we were using all medically available information to characterize causes of death, and this had us attribute more deaths due to asbestosis than would have been attributed by McDonald, who used death certificates only.

Q. Whereas you used...

A. We used autopsy protocols and medical records

A. (cont'd.) as well as death certificates, for the attribution of death.

Q. You say...

A. Additionally, there may be something different in our group in terms of the age distribution as well, although with his I was using the twenty-plus year group, as well as in our case we had a twenty-plus year group. But his twenty-plus year group was a different one in that their exposure was to asbestos in different calendar periods of time as well.

Q. Before you lose me completely, I would like to go back to your first answer, if you don't mind. Talking about using Asbestos, which had the lower mortality rate or SMR, than in Thetford, which would explain the difference...

A. It doesn't explain it all. I mean...

Q. It could be part of the explanation, that's what I understood you to say.

When you did your own study in Thetford, you did not study Asbestos, the Asbestos workers?

A. Right, and we only studied part of Thetford.

Q. Yes. In fact when you studied Thetford, you studied the employees from Asbestos Corporation?

A. Yes, the company that is now so-constituted.

Q. They had the greater number of employees?

A. Right. I'm not...we had Carey in there as well...

Q. As Quebec Asbestos?

A. Right. There was a couple of other small mines, and I forget whether they were in or out of the Asbestos Corporation complex.

Q. We assume that you studied those mines which were CSN affiliated?

A. That's what we did.

Q. And that included Asbestos Corporation, which had the greater number of employees?

A. Yes.

Q. And it may have included Carey, which I think was CSN, and maybe National, which may have been CSN at the time? Which would make it about three mines, excluding Bell Asbestos, which was methylose?

A. That's correct. It excluded Bell.

Q. And Lake Asbestos?

A. Yes.

Q. All right. Now, did you have occasion, Dr. Nicholson, while you were there in Thetford, to compare the working conditions in Asbestos Corporation with the working conditions in, say, Lake or Carey or National?

A. No. Unfortunately, the companies were unwilling to provide a tour of the facilities for us, which has been in contrast to other mines and mills that we have studies.

Q. You were dealing with the representative of CSN...?

A. No, we wrote to the company as well, and asked for their co-operation.

Q. But Dr. Nicholson, I'm not, you know, the Corporation is not one of my objectives. What I want to know from you is, in speaking with the union were you able to get a handle as to in effect what would be the working conditions in one mine as opposed to the other, because of age, if only?

A. The workers described some of the conditions in the mines over the years. I don't have those detailed descriptions with us, but we had conducted considerable interviews with workers on changes that took place.

The measurement...and indeed different mines had different typical air concentrations. We found in our measurements, even, that one mine...I think it was the Carey Mine...

A. (cont'd.) had considerably higher concentrations than did the main mines and mills of the Asbestos Corporation.

Was it the King Beaver that was the main complex?

Q. King Beaver was the main ...

A. The main...right. Than the King Beaver complex.

Q. Dr. Nicholson, did you have occasion to know from the workes what the working conditions were at Bell Asbestos?

A. No.

Q. What about the working conditions at Lake Asbestos?

A. No.

Q. I'm going to suggest something to you to see whether it can perhaps work.

If I suggested to you that Lake Asbestos was a fairly new mine at the time, that the equipment was quite modern, and that indeed at that time it was a very clean mine where the dust concentrations were very low...if I suggested that to you and added that it had, at that time, over three hundred and fifty employees...all right?...if you had added to your cohort these employees, would it not have changed to some extent the result of your study?

I'm going to finish with this...in the same manner as you say the workers from Asbestos could have changed it because of the low mortality rate, albeit Asbestos having a greater number of employees?

A. No, no. First, I'll go back to the Asbestos/Thetford comparison. The understanding that I had with the discussion in McDonald was that per estimated exposure, the differences existed. It was acknowledged that Thetford had higher dust concentrations than did Asbestos. But separate from the fact that it was higher, the relative effect per unit dose was greater. So McDonald was looking at, was comparing effects

A. (cont'd.) at given exposures, and he found different effects for the same exposure category in Thetford, compared to Asbestos.

Now, going to the conjecture that you made of including Lake Asbestos, if we had done so in our study, and the numbers of people were dramatic...were large and equalled those of Asbestos, and we had the available air measurements that would indicate that first, conditions had remained low over years, and taking that into account where you would then have reduced the average exposure to the group that I would have estimated.

Q. Oh!

A. So the effect per unit dose could be the same. I don't know if it would be, but you would take into account the air concentrations of Asbestos that would be present, to the extent possible.

Q. I think what you are saying to me is that given your theory on averages, it wouldn't have made any difference?

A. It might not have made any difference.

Q. It just goes to show that what's the sense of keeping a clean mine, eh?

A. No, no. You would have seen a reduced mortality from a reduced dust exposure, but you could have gotten the same relative risk, and that's what's counting. By all means keep a clean mine.

Q. Thank you.

Dr. Nicholson, you, I think in tab eight, yes, it is tab eight...this is your study on miners and millers in Thetford Mine...

A. Okay.

Q. ...and when I look at page one thereof, I see that the cohort was established of all men employed during 1961, with at least twenty years of seniority in any one of four

Q. (cont'd.) companies mining and milling. Could you give me the name of the four companies?

A. I'll try to. I mean, they came from the records of the Asbestos Corporation, which had their King Beaver seniority list. They also had a seniority list from the Johnson Mine, I believe...that name comes to mind...I mean, I've got the records back at Mount Sinai...which was then incorporated into the Asbestos Corporation and no longer was carried...in later years was not carried as a separate entity.

Q. Okay, I think maybe I understand. What you are saying, in effect, is that in point of fact, legally speaking, because that's what I read, you had two mines - you had Carey and you had Asbestos Corp?

A. I think we had National, too.

Q. That would be three mines.

A. I think so.

Q. But you say...

A. At the time of the records, it was four.

Q. Four companies? Individual companies?

A. Right.

Q. It would have been what, Asbestos Corp, Carey and National? That makes three.

A. Johnson...or B.C., what does that...?

Q. I know it's been a long time, but perhaps you and I can agree that when you said companies here what you really meant was sites? Maybe that's what you meant? Because in effect, Doctor...I mean, I don't want to tangle with you on this...I know more about the legal side than you do. It wouldn't be fair to you.

A. I'm not trying to make a big issue about it. We had records from what were four separate...from what I understood to be four separate corporate entities in 1960...or what was it, 1961? Yes, 1961. One of those was known as the

A. (cont'd.) King Beaver Mine at that point. I don't think Asbestos Corporation was even a corporate entity at that time, was it?

Q. No, I think it was. I think what you had at that time, you had Asbestos Corporation, you had Carey, you had Bell, you had Lake, you had National, and perhaps another one.

A. There's two names that I recall, and I don't know when they were combined into Asbestos Corporation...are something called Johnson and B.C., and I forget what B.C. stood for.

Q. I know what it's for. We'll leave it at this. Perhaps we can proceed otherwise.

It's just that I see here that you had in your introduction here, you talk 'all men employed during 1961 at least twenty years'.

A. Right...on those seniority lists.

Q. Yes, all men employed with twenty years.

A. That's on an hourly seniority list. It does not include supervisory personnel.

Q. The seniority list was handed to you by the union?

A. Yes.

Q. They brought the people to you who were supposed to have twenty years experience, or you checked the list yourself?

A. No, no, no. We got the lists. Nobody brought the people to us on this study. We hired someone to call around and identify who was alive and dead. I mean, this was a mortality study.

Q. Yes?

A. We had the list, and we had all these names. The lists were identified by a clock number or something like that, as well as by the date...his name and the date that he

A. (cont'd.) was first employed by that particular company.

Q. All right.

Dr. Nicholson, I seem to be a little mixed up, and I think it's because I'm perhaps confused with the fact that you went twice to Thetford for a study, did you not? You went once for a study which is referred to in this tab eight? Right? Tab eight, which is entitled, Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec. Have you got that?

A. Yes.

Q. That's one study?

A. That's correct. But that study was going on over a period of months.

Q. Yes.

A. In fact, it was going on over two periods of several months each - once in the 1973/74 period of time, and subsequently in the 1977/78 period of time when we followed up again the group that we had identified initially.

Q. Well, Dr. Nicholson, referring back now to the Beaudry Commission hearings, and do you not recall that sometime prior to those hearings, at the time of a strike that was taking place in the Thetford area, you went around there to make a study. Now was that a mortality study or a morbidity study, or what was it? Your whole team went down.

A. Yes. We went...a large group from Mount Sinai went to Thetford Mines prior to the strike...they were not on strike at the time that we went, and that was to conduct a clinical survey looking at the prevalence of abnormalities among largely twenty-year employees of the Asbestos Corporation at that time.

Q. Were you then checking your records or updating your records from the previous study?

A. No, I don't think we were doing anything at that time about the records of this study. Those records also provided, to the extent that we had already virtually completed this study by that time, and were using those individuals...we invited those individuals that were retired from the list that we had established here, to the examination as well.

The examination included roughly eleven hundred currently-active employees with more than twenty years seniority, plus about one hundred or one hundred and twenty-five...I don't remember the exact numbers...who were retired from mining and who were identified by virtue of having been on these lists in 1961, with twenty years seniority. Many of the 1961 people were still active, and thus were invited because of that. But those that had retired were also invited.

Q. What I want to know from you is this, Doctor, I'm a little mixed up now. That was in 1975, or thereabouts?

A. It was November, 1974.

Q. Okay. Is that the time when you were examining something like twenty-five individuals per hour?

A. No. Well, the number that were going through were two hundred and fifty a day, but the day was a lot longer than ten hours.

Q. I suggest to you that you were doing twenty-five individuals per hour.

A. Between twenty and twenty-five.

Q. Were those x-rays?

A. They were x-rayed, ten physicians were performing chest examinations, two stations were doing pulmonary function.

Q. I don't want to get you mixed up, so perhaps I should refer you and have you read your evidence before the Beaudry Commission, at page thirty-four...if I may

Q. (cont'd.) hand it to you?

A. Sure.

MR. LASKIN: I think...

THE WITNESS: Should I have it?

MR. LASKIN: You did.

THE WITNESS: Right, I've got it.

MR. CASGRAIN: Q. You've got another copy?

THE WITNESS: A. Yes, I do have a copy. Wait a minute. It's right here. Let me get it.

Q. Page thirty-four.

A. Okay.

Q. Line twenty and following.

A. Yeah, approximately twenty-five per hour. We were doing two hundred and fifty a day, but some days ran a bit longer than ten hours, is all I'm saying.

Q. Oh, I see. That's an average? Is this an average?

A. Well, this was the intake rate. Instead of finishing at seven or eight o'clock, we were finishing at ten o'clock.

Q. My question to you is the following: Were you at that time updating your records?

A. No.

Q. What were you doing?

A. With that many people coming in, we weren't doing a damn thing about records. We were trying to determine the prevalence of asbestos-related disease among a population of twelve hundred and fifty workers.

Q. Is that the occasion, Dr. Nicholson, when you said that you had found to your horror and dismay that there were more than thirty percent of the group examined who had developed abnormalities qualified as zero one?

A. No, it was a lot more than that.

Q. Oh, is that right? Perhaps we will find in due course...

A. Oh, I'm sorry. Thirty percent had zero one?

Q. Yes.

A. Overall?

Q. Yes.

A. Or less?

Q. Overall.

A. No, zero one or less overall? I'm sorry. Repeat your question again.

Q. Is that the time when you said you had found, to your horror and dismay, that over thirty percent of the people examined by you had anomalies qualified by being zero one?

A. No, I would not have made that statement.

Q. No? Will you try and recollect?

A. Let me say what we found, because I don't know what...of the group that we examined, the overall percentage of abnormalities of one slash zero or greater, was of the order of seventy percent.

Q. What about zero one?

A. Well, zero one or zero zero would be the remaining thirty percent, and those would be categorized as normal.

Q. Did you eventually publish those findings?

A. We presented them to the Beaudry Commission.

Q. Did you publish them anywhere?

A. They are not published otherwise.

Q. They were not published anywhere else?

A. At this time that is correct.

Q. Why were they not published anywhere else?

A. A variety of reasons. For one reason, they

A. (cont'd.) never got written up by the physician that was directing the laboratory at this time, because of pressure of other activities.

Q. So we will never see what the results of this was?

A. No, I would hope that you would see them.

Q. Sorry?

A. I would hope that they would be available in more widespread form than they are.

Q. In the course...did you use the same standards for your examination during that particular venture that you did when you were carrying out your other mortality epidemiology study, which is tab eight? The same standards?

A. The same standards?

Q. Yes.

A. They were different types of studies entirely.

Q. Oh, is that right?

A. One was a clinical survey, one was a mortality study from records.

Q. In using the number of employees, or the employees to whom you refer, is it the same makeup with respect to the companies? Was it the CSN group?

A. Yes, it was the CSN group.

Q. Was it approximately the same number of people?

A. No.

Q. Oh, no. Because one was clinical and one was mortality, is that right?

A. Yeah, one was identified in 1961, and one was identified in 1974.

Q. One last question on this particular score, Doctor. Is it possible, perhaps, that one of the reasons why

Q. (cont'd.) there are differences between your study and that of Dr. McDonald's and because your percentages are higher, is it possible, perhaps, his cohort was more representative than yours? I'm talking about possibilities, without ascribing any blame to you.

A. No. He had certainly a much greater number of individuals, and thus variabilities associated with small size which we certainly would have had. Because the number of deaths that I reported on are, what, a hundred and ninety? A hundred and seventy-eight.

With that, the variability in the number of deaths by cause is considerable, so that in that sense the larger study is a more representative one in the sense that the variability of the numbers would be less.

The issue, though, still exists of systematic differences that could exist between the two...the use of the... not considering asbestos disease unless it was characterized on death certificate, and the problems that come from ignoring deaths that should be attributable to the exposure, the problem that comes with the difficulty of translating particle counts in a variety of circumstances to fiber counts. Such systematic differences can considerably outweigh statistical differences, and to the extent they would or would not is an issue that people could debate from here to midnight.

Q. Tell me, Dr. Nicholson, you describe somewhere in your writings, and I think it was not mentioned by you verbally, what you consider as best evidence. Am I correct in saying that when you say best evidence, we see BE on your tables, you are referring to having used autopsy rather than death certificates? Is that what you call best evidence?

A. The best evidence would be that...the best evidence...there is a hierarchy. An autopsy where you look both at macroscopic and microscopic specimens after death would be

A. (cont'd.) the best evidence that would relate to the cause of death.

Next is surgical specimens and medical records, clinical records of the individual at the time of his terminal stay in a hospital.

Finally, of least validity compared to those, would be the record as manifest on a certificate of death, which could have been signed by some doctor that just sees the guy dead and has no awareness of any previous history of the individual, and often it is written off as a coronary when in fact other causes of death may have been in fact related to the...

Q. You checked all of these points for every single case?

A. That's correct. We checked as much...some of them we used only death certificates because that was all that was available. But for those individuals for which other information was available, that was reviewed. We reviewed all the records available in the local hospital for everyone in our cohort.

Q. How many...what's the percentage of autopsies that you had available?

A. The number of autopsies is about fifteen to twenty percent, if I recall.

Q. Does that come from the Workmen's Comp or from the local hospital?

A. The local hospital. The local hospital.

Q. The local hospital?

A. Yes.

Q. Did you speak to the doctors who had performed the autopsies?

A. Yes, largely so. It was largely done by one...Mark Poulin was the one who did most of the autopsies.

Q. Did you, when you began...

A. I did not myself review those protocols. It was done by a physician.

Q. When you went down to carry out your study, the one that has been published, did you know that Dr. McDonald was carrying on a similar study, or had carried on a similar study?

A. Doctor who?

Q. McDonald.

A. Oh, I knew that he had published a study earlier of the mortality of miners and millers.

Q. I find it a little surprising, and perhaps you can correct me if I'm wrong, is that throughout your evidence you have taken various reports of various people, and you have... not that I blame you, it's your business, not mine...and you have from those extrapolated and drawn conclusions...and you have even said to us in cross, that dose response was perhaps not that material, averages were better? In fact, yours is better than McDonald's because it doesn't have that sort of a bunch of facts in it?

Then you decide to carry on a study in Thetford, and instead of going to Dr. McDonald, who has all the data and would have provided you with the data from which you could have extrapolated, you decide to go down on the scene and there you go to the trouble of having best evidence by examining and targetting the doctors, looking at the hospitals, looking at the records, and examining every autopsy to make sure of that. Were you trying to make sure that you would find more cancers than Dr. McDonald, or what were you doing?

A. We were trying to...

Q. Because I don't see...I'm not finished...I am trying to see why in this particular case you were so concerned about the facts and why you were so little concerned about the facts when it comes to analyzing and extrapolating. Would you please give me an answer to that?

A. We were trying to find out what the causes of death were, accurately. So that's why we were doing that. On the extrapolations we were trying to utilize the best data available for those extrapolations. Extrapolations that I've made, did that.

Q. You didn't want to rely on Dr. McDonald's figures, is that it?

A. At the time that it was published, it was not possible to make estimates from Dr. McDonald's data of what would be a dose-response relationship for much of the disease experience in those mines and mills.

Q. Did you ask the McDonald group for the figures?

A. They did not have data in 1973, other than... that allowed dose-response relationships, other than for lung cancer, which showed that there was a considerable excess of mortality in that group that he studied.

Q. Would you go back to page ninety-eight of your transcript of your evidence before Beaudry? Dr. McDonald, Alison McDonald, is asking you a question there. It's in French and I'll translate it literally as I go along.

A. Yeah, you better, because I don't...

MR. LASKIN: I'll have the chairman check up on that.

MR. CASGRAIN: Q. But there was official translation there.

THE WITNESS: A. Oh, well let me read...is there official...where?

Q. No, no. It doesn't show that it was translated, but it was. I'll read you the question here at the bottom of page ninety-eight, line twenty-five.

"Dr. Alison McDonald: My second question is more general..."

This is Dr. Alison McDonald, Dr. McDonald's wife.

A. Yes, I know.

Q. "How is it that while knowing that we had carried out vast researches in Thetford Mines since 1966, why the staff of Mount Sinai came to Thetford, carried out researches in 1974, without telling us and without asking us for our own information? This could have negated the possibility of two different stories on asbestos appearing. We now find that our results look the same."

Now, that was a question put to you by Dr. McDonald, and you have your answer there.

A. Yeah, but the results don't look the same. Can I read my answer?

Q. Yes.

A. "If they were the same, that would be in fact good." I'll read exactly. "I'm saying that the results were considerably different." My answer then: "If our results are parallel and similar, and in fact identical, then it is to the benefit of all. We did have contact, prior to the survey, with your group. We met with two Canadian physicians and with Dr. Becklake, who was the only person from that group we were able to meet with, prior to even establishing the fact of the survey, when it would be held or making any arrangements for it.

We wrote to the secretary of the Asbestos Corporation asking if he were willing to co-operate. We assumed that he would have contacted your group since your study had been conducted using company films, but no co-operation was forthcoming from that industry

A. (cont'd.) "source, and in fact no co-operation was offered by Dr. Becklake. If the McGill group and the QAMA group had been willing to co-operate with us and provide data, they had ample opportunity to do so."

Q. And to volunteer it to you rather than wait for it to be asked for, is that what you are saying?

A. Yes. If that had done so, we would have been happy to co-operate with them in any way...

Q. And you would not have carried out all these specific studies if we had said to you, look, you can look at McDonald's studies? You would not have gone on to Thetford? You would not have checked the medical certificates?

A. No, the medical study that we did was considerably different from what was done by the McDonald group. It was a larger group that was surveyed and for which x-rays, pulmonary function and clinical examination was conducted. That is, they looked at all the x-rays of everybody that had been employed at any one time, and read those x-rays and did that, but they did not do that on a currently-employed group and they were using x-rays that sometimes had been taken twenty or thirty years ago.

Additionally, we provided the information on the results to the union involved, which was not done by the people. Not a single individual that they saw ever received a report of what the findings were in his particular case.

Q. Perhaps the last question is somewhat stupid, but I'll try it anyway, Dr. Nicholson. From what you are telling me now, the McDonald study and yours are very different?

A. No, they are not very different. I'm saying they are different. We found a considerably greater prevalence of x-ray abnormalities among the population at Thetford Mines

A. (cont'd.) than did McDonald.

Q. Doctor Nicholson, it seems to me that what I asked in the beginning was the following: Why did you bother to go down to Thetford when you could have had the data from McDonald, all right? You said finally you went down and didn't ask for it and you were not offered it, therefore you carried on.

Then I asked you why it is that you did not take simply the data to extrapolate like you do in all your work. Then when I pressed you further, you said 'we proceeded in a different way'.

A. Wait a minute. You are asking...I'm not sure what studies you are asking the questions about. When you are talking about the analysis of data and extrapolations that I have used in this tab nine, the only data that appear here that was used for estimating cancer mortality were data on mortality studies. We conducted a mortality study, McDonald conducted a mortality study. The results of each are included here.

Now, the issue of the clinical examinations that we conducted is a different one, and has nothing to do with this report at this time.

Q. Okay. Fine. All right.

A. The findings that we had in each of these examinations are somewhat different. We find a greater...the results that would be obtained using the data that we had on the mortality study of the group of workers employed at Thetford and organized by the CSN has percentages of asbestos disease per fiber exposure, per fiber dose, greater than that of McDonald.

They are similar in the sense that compared to other studies, they are much less than, for example, factory employment or insulation work. Together they are less.

When we looked at the clinical status of the

A. (cont'd.) workers, the prevalence of abnormal x-rays was also greater than that which was reported by McDonald.

The differences were largely in the categories of the number of x-rays categorized in category one. There was close agreement between the number of x-rays categorized in catgeory two and three on the ILO scale.

Q. What's category one?

A. Category one on the ILOUC scale, the broad category.

Q. Zero one?

A. One.

Q. One.

A. That is, it should be one zero, one one and one two, but I don't remember the breakdown for each subcategory.

We offered these x-rays to any groups to read, that we had taken. To any group to read, including the British panel of twelve readers. Dr. McDonald said that he did not want that group to read our x-rays, and thus that...

Q. Did he tell you why?

A. He didn't tell us why. We were told by the British that he had objected to their reading our x-rays.

Q. But he has never told you personally he didn't want them to read it?

A. He told them that. We sent them to the University of Pennsylvania to be read. In contrast to our findings, seventy or seventy-five percent abnormal x-rays, Pendergrast found more than eighty percent or eighty-five percent were abnormal. He found at least a ten percent greater number of abnormalities.

And this is in contrast to what McDonald was reporting on a similarly-categorized group, in which he found

A. (cont'd.) something of the order of thirty to forty percent would be abnormal.

Q. Was he biased?

A. There can be biases that can develop in the reading of the x-ray. In particular in his group of x-rays that were available to him, the quality was not particularly good for many of them. They were often taken many, many years ago and both film deterioration and the quality of the x-ray at that time was not as desirable. We had a very uniform quality which is of high quality, and McDonald acknowledged the quality as being much superior to that which he had available to him.

Q. When and where did he say that?

A. When he visited with us.

Q. When? What date?

A. In the seventies some time.

Q. What year? What month?

A. I don't remember the month.

Q. You are quoting Dr. McDonald, Dr. Nicholson, as if everything...

A. I can quote him because I remember what he said. I don't remember the date that he visited Mount Sinai.

Q. Do you remember the year?

A. It was probably 1975.

Q. Or 1976?

A. It may have been.

Q. Or 1974?

A. No, it would not have been 1974, because we only took the x-rays in 1974.

The other aspect is that most of the x-rays that were being read in his study were normal because they were people that had been employed for short periods of time, or only recently. Thus, because of the way it was constituted, because

A. (cont'd.) of the very large number of such people in it, the x-rays were still normal. Thus, as one sees a whole bunch of normal x-rays coming by in a reading procedure where you are reading very, very many of them, ones that are marginally abnormal tend to be read as normal. So there is something called a field effect in the reading, that takes place, that could have influenced his reading.

By his reading, I mean the readings of the study that was...

Q. That couldn't happen to your group, the field effect?

A. It did not, I think, happen to the same extent.

Q. How do you know that?

A. Because if I...

Q. It's quite subjective, isn't it?

A. Pardon?

Q. It's quite subjective. How do you know that?

A. From the comparison of the readings that have been done by others of those films, by the use of those films when admixed with other studies. And it was not found.

Q. Dr. Nicholson, you just said that as they were reading, because they were reading a great number of normal along with abnormal, they would have a field effect and therefore be influenced. That's a very subjective way of doing it, is it not?

A. Yes, it is. But reading of x-rays is very subjective.

Q. It's just as subjective for your group as it is for McDonald, is it not?

A. The field effect was less present there because we did not have the vast number of normal x-rays as the background.

Q. So now we have it that if you have lesser x-rays, you have a better result than if you have more x-rays? Is that what you are saying to us?

A. No, I wasn't saying that at all.

Q. No?

Did you...how did you proceed to do the reading, in effect? What technique did you use?

A. We have five readers...four readers read each x-ray individually, and then they were all read with the individual readers having available their reading, read again as a group, and a consensus reading derived.

Q. Would you tell me where the x-rays came from?

A. They were taken by a portable commercial x-ray unit.

Q. The x-rays you read were all the x-rays that you had taken with this apparatus?

A. Yes.

Q. Dr. Nicholson, did you and your readers who sat and read those x-rays, then wrote down their results, is that right?

A. Over a period of time, yes.

Q. You had, what, five readers, six readers?

A. Four, I believe.

Q. Four readers. The four readers, did they cross-read the x-rays? Or did they read them just once? Each had a bunch of x-rays?

A. No. Everybody read a portion of the x-rays a second time.

Q. The same x-rays?

A. Yes. So another group was admixed into them.

Q. There were no other x-rays involved?

A. On the initial reading there were no other

A. (cont'd.) x-rays involved.

We have also used these x-rays with other x-rays.

Q. When?

A. Admixed...

Q. The second time?

A. Yes.

Q. The first time you didn't use other x-rays?

A. That's correct.

Q. Why?

A. Because the initial readings were made in order to provide a report to the individuals as rapidly as possible, and we did not include other x-rays in that study, on the initial reading.

Q. When you say you included other x-rays in the second time of your reading, what other x-rays did you use?

A. I don't remember. We have read the x-rays at other times with a variety of other groups. I do recall the x-rays being read, I think in conjunction with our study of the mining population in Baie Verte, but I am uncertain as to...

Q. With the Asbestos workers as well?

A. Yes.

Q. What I want to know from you, Doctor, did you at any time introduce in the x-rays you were reading, neutral x-rays? That is, x-rays from workers who had nothing to do with...?

A. Yes, we did in another reading of those x-rays, is what I'm saying, and the reading...

Q. The second time?

A. Yes. And the readings were exactly the same, in fact even higher, than they had been read with the initial reading. So the estimates that we had made, in fact, from all subsequent readings by ourselves of those same x-rays,

A. (cont'd.) from all readings of anyone else were an underestimate of the prevalence of abnormalities, and at this time if the British panel would like to read those x-rays, they are welcome to do it, or any other recognized panel. We would be quite happy to have our readings compared with any other recognized reading group.

Q. Just to make sure we are not making any mistake, are we now talking about the x-rays taken on that visit to Thetford in 1974...?

A. Yes.

Q. Yes?

MR. CASGRAIN: I have no other questions, Mr. Chairman.

DR. DUPRE: Miss Jolley?

MISS JOLLEY: I feel that we had agreed that the Commissioners would have their hour, and it looks like we are going to go over.

DR. DUPRE: For the time being, be the Commissioners' guest, Miss Jolley.

MISS JOLLEY: Okay.

CROSS-EXAMINATION BY MISS JOLLEY

Q. Dr. Nicholson, I wanted to talk about the underdiagnosis of disease. We dealt with that slightly yesterday, and you went through, in the discussion of Selikoff's material, about insulation workers. I think Dr. Uffen asked you yesterday that we had a presentation before the Commission from the insulation workers, indicating that Dr. Selikoff had found in fact about thirty percent...or a third of the death certificates were changed, with the best possible evidence, following investigation. Perhaps a third of the...?

A. Well, a third of the...I'm not sure if it

A. (cont'd.) wouldn't have been a third overall. There would be fifty percent more mesotheliomas added because of that investigation, and that is one-third of the mesotheliomas were misdiagnosed.

Q. Right, were misdiagnosed.

A. And at least a corresponding number of asbestosis.

Q. Right, right.

A. But for other causes that percentage would be less.

Q. Right, right.

I think we are really concerned about this whole issue of underdiagnosis just because of the effect on calculation of risk, and also exactly what the doctors are doing.

Last week, with Dr. McDonald, Mr. Warren pursued a line of questioning suggesting that perhaps the physicians in Thetford area in Quebec, would in fact probably be more sensitive to the issue of asbestos disease, and therefore perhaps would overdiagnose that disease.

Now, Dr. McDonald, in response, indicated that he didn't feel that was necessarily true. But clearly when you investigated in your study, you found that there was significant underdiagnosis in that area.

A. Particularly for asbestosis.

Q. Yes.

A. We did not...we found one case of mesothelioma. I don't remember whether that was diagnosed or not. So I can't say for that.

Q. It's of concern to us because I think that the asbestos people have been trying to maintain that perhaps their records are better, and I think that underestimation of death may have an impact on what Dr. McDonald is finding as well.

I want to pursue another...

DR. MUSTARD: Counsel...or not counsel, I realize, but can I undertake a question at this point?

MISS JOLLEY: Sure.

DR. MUSTARD: Because I'm going to ask it anyway.

MISS JOLLEY: Be my guest.

DR. MUSTARD: Table one, this is the best evidence for the information? This is in tab nine?

THE WITNESS: Yes, right.

DR. MUSTARD: This is the final compilation of all that data, because I think when we heard testimony it was inferred that it was being completed, but it had not been completed. What I would just like to have recorded is, have you now done the full review of all the insulation workers in terms of best evidence versus the death certificates?

THE WITNESS: Oh, this table one...

DR. MUSTARD: Have you got a study in process that's going to...

THE WITNESS: No, no. This is a study that characterizes the results of the mortality of the workers through 1976, and of that group that had died to 1976, the entire review has been completed. But the study is a continuing one and the mortality experience in subsequent years is being accumulated and will be reported at a later time. So there will be presumably some publication in the future on the mortality experience of workers from 1967 through 1992.

DR. MUSTARD: I guess I was left with the impression that there was something in the mill that was coming up on this subject, but I take it that there isn't, in your organization?

THE WITNESS: Well, there is considerable research being done with these people, looking at other aspects of asbestos disease. But in terms of these data, this is complete for the period of time that it is stated to be the case.

THE WITNESS: (cont'd.) There is not going to be any alteration of such data, and because the numbers are so large, with a hundred and sixty-seven thousand person years at risk, future data will not be that different. But you, of course, can have statistical variations and maybe some age effects will be manifest in later years, but this accurately reflects the situation as it is now and like will be.

MISS JOLLEY: Q. It is true that when you underdiagnose disease then you would be underestimating the risk when you carry out your studies using the death certificates?

THE WITNESS: A. Yes, if you limited yourself to only a specified few diseases, as previously recognized.

On the other hand, if you underdiagnose on one cause, then there will be a correspondingly greater number in some other cause, and...

Q. And you dealt with that yesterday when you described the pancreatic cancer.

A. Yes, and you have that problem as well.

Q. That kind of thing, right.

I think the one thing I want to make clear from yesterday's presentation in the morning, because it was quite complicated and new, was that Dr. Enterline, when he was before us, made the proposition, I think, that was presented yesterday, but I just want to make it clear. The time to tumor that Dr. Enterline presented was that there is a dose latency situation, and that the lower the dose, the longer the latency. Therefore, what he was proposing to us is that you could set an occupational standard low enough that the time to tumor was in fact longer than the average lifetime of a person, so that the person would die of something else before he would get a chance to die of cancer. What you presented yesterday seems to say that that's not true. Could you just...?

A. Yes, I would very strongly say that that's not the case. For example, I think what Enterline has utilized is that time to tumor, which in principle would be a mean time to tumor, would be inversely related to one-third of the dose. That is...I don't know if it's appropriate to go through it again, but that is simply a manifestation of the reduced risk associated with that reduced dose. The probability of cancer being less, the number of cancers reaching a level of significance, would appear at a later time.

In terms of...and I'm sure if I discussed this with Dr. Enterline, he would agree with that. Some things one can...you know, neither McDonald nor Enterline are malevolent people, and McDonald and I could argue, for example, about whether this way of analysis or that way of analysis is more representative of the case, and it would not be resolved because we would be saying that would be a subjective judgement as to whether average exposures that I would propose would best represent the dose that these workers would have, or those that he calculated. Those judgements are subjective and not easily resolvable.

But in this issue, if I went over it with Enterline I'm sure he would agree with me, because it's unequivocal from the data that exist. When one translates them into dose-response curves, the dose-response curve that you see is exactly that which would predict a time to tumor equal to one over the cube root of dose, or achievement of a certain percentage of tumors versus time.

But it's a dose effect and not separately a manifestation...if you understand. I'm not trying to be...I'm trying to separate judgements in terms of what one would argue about.

Q. I just want to be clear because I was confused.

Q. (cont'd.) Some evidence that came before us not last week, but the week before, Dr. Weill was here and presented his paper. I was a little confused because he had a twenty-five percent part of his group that were not traced and that he in fact presumed alive, as part of his cohort. At the same time he had extremely low SMR's in his material, which would indicate that there was a potential bias in his inability to followup on the twenty-five percent.

However, in trying to explain it, and this is where I am somewhere at a loss, he said that all the deaths would have been distributed equally and it didn't really matter. I just wondered if you could tell me whether I should be concerned about a twenty-five percent lack of followup?

A. Yes, I think there is a real important concern there because, firstly, the results where you have not... where you utilize only a portion of the deaths certainly distorts the overall picture, and the finding that he has an SMR of something like fifty or sixty percent in some categories, suggesting that one of the healthiest places a person could go to is an asbestos factory, is false.

The reason that the number of deaths are underestimated is that when one uses social security records for tracing, prior to 1967, particularly, there was a lack of knowledge on the part of the Social Security Administration of the United States to identify deaths of people who had previously contributed to that system if there were no survivors alive at the time of death that claimed a benefit. At the time of death, people were aware of survivor benefits...that is, to a widow or to dependent children, but if neither, an individual died without those being available, often a small death benefit, a small burial benefit was not claimed, and unless it were, the Social Security Administration did not know that the guy died. For their purposes it would be an inactive file.

A. (cont'd.) In Enterline's case, twenty-five percent of the individuals had inactive files in the Social Security system. Some of these could be because they in fact were not working at that time and not receiving money from them, but it also could be that many of them were deceased and unknown to the Social Security system.

The people that are most often...may have been in this category could have been those that were...they may or may not have been equally distributed amongst the different categories that he subdivided his group into. If they were not distributed, then even dose-response relationships, even the slopes of lines could be in error. Unless one has that proper information, one can neither make estimates of the overall effect on the group, or of the trend with estimated exposure.

Q. Okay.

I want to pursue a line of questioning based on your paper on TLV's as a concept for controlling carcinogens. I don't want to focus on that exactly, because I think you have made that quite clear, but the thing that we are concerned about in the labour movement is, when you set TLV's, or time weighted averages for any substance, but more importantly for a carcinogen like asbestos, our TLV's or time weighted averages in Ontario, when we do get any standards, which may be in the fall, are going to be based on a forty hour week, not even an eight hour day, which means that...and presumably will have some ceiling measurements...but you are going to have situations where workers can be exposed to fairly high concentrations of fibers for X number of hours a day, but when it's measured over a forty hour week you are going to in fact have time weighted averages significantly below the TLV, and therefore not in violation of law. But the high intermittent exposures that those workers are going to get from those high, short-term things are going to be a significant health risk.

Q. (cont'd.) I mean, we are concerned really not only that TLV's are inappropriate for carcinogens, but the way...they are even measuring over a forty hour week, which gives us even less protection.

A. Yes, I think the problem you point to is a very real one. In fact it is the essence of control for asbestos in the future, because it comes about that the peak exposures are particularly a problem in the asbestos-use industry as opposed to the asbestos-manufacturing industry. That is, they are not...occasionally, of course, in a factory something breaks down, but in general that is a more standard and evenly-controlled circumstance. But the exposures in maintenance and repair, and in construction work where asbestos is already in place, are largely going to be those of the peaks. If you can eliminate those peaks and deal with those, and focus on how to minimize that peak exposure, you can largely control the problem.

To try to deal with a time weighted average of forty hours, in essence says that it's not the problem of the peaks we have to deal with, it's this nebulous forty hour exposure that is impossible to determine for a construction worker undertaking boiler repair work one day and water tower repair work the next, and bathroom leakings the third, and then back down to putting a fixture up in the crawl space, plenum space of a building. So I think you really want to have control measures designed to deal with that particular problem especially.

Here, I strongly feel that with the problems that exist...that will exist in the future, work practices are an important aspect of any regulatory principle. You can apply TLV's to indicate where you feel standards that should be achieved, but where either work practices can be applied effectively and peaceably that would achieve more average exposures, to, in essence, eliminate those peaks in the

A. (cont'd.) construction industry, those should be applied. One should not, in essence, attribute a numerical standard as something...as a TLV for asbestos as being something that's safe. It should only be utilized to, in essence, rule our processes that can meet it, and that the guidance principle should be one of eliminating, to the extent feasible, all unncessary exposures, which has been talked about previously.

It's going to be in maintenance and repair, and that's where one really has to look at how to deal with that. It's a very difficult problem to deal with.

Q. I guess following on that is the whole issue of the schools program that's going on this summer in Ontario, and you talked yesterday about the importance that companies have experience in the removal, that it both shortens the time, plus it makes it a safer operation. I think that...I guess what I would like to pursue with you on that is that, how do you control these contractors? I mean, we've got a lot of contractors that are moving through this business very fast, and the scary thing is they are picking up kids, probably, this summer and putting them into these programs, with no records and no controls. How do we go about trying to control that kind of operation that may be going on?

A. The only suggestions I have, and in part it comes from some experience in New York City that attempted to operate in this way, is that if a contractor wishes to enter the business of asbestos removal, he should first demonstrate his awareness of the problems and demonstrate the adequacy of his techniques prior to being, in essence, certified to bid, or something like that. So there can be at least an initial screening that basically would be an evaluation based upon plans and demonstrated knowledge of what the problem is and a description of the techniques and equipment that he would use, that would allow an evaluation to be made.

A. (cont'd.) The ultimate result is what he does in the field, so that once allowed to bid with criteria that have been properly established, then he would maintain his license to bid on jobs only upon the maintenance of what are determined to be safe work practices, so people could monitor his work activity and if it is in compliance with a code of practices that indeed protects workers, indeed seals off the building and eliminates the dissemination of materials and fulfills all the desired attributes that everyone could wish to achieve, then he can continue to bid. Anybody can enter that without...it's not a prohibition for people entering the business, but once in it they have got to maintain those standards, and if they don't, they go out.

Q. So if you move to a licensing of contractors to do this kind of business you would also have to have very stringent enforcement or overseeing capacities within your licensing agency?

A. Right. You would not have to have somebody on every job. You would, in essence, be monitoring a contractor depending upon what his previous...the previously-observed activities were. The first job that a guy does, you might have somebody there the whole time and he might have to take into account that this is his first job and do as we ended up doing, helping the guy out. If he's really trying, that's okay.

But thereafter, he's got to produce. As contractors continue to produce, then you monitor them less and less. So it isn't an onerous observation job. In fact, the number of jobs at any one time are fairly limited, so we are talking about, in a given geographical area, perhaps two or three people as the asbestos inspectors, in this scheme.

In New York City they had a guy that...Tony Smith and a couple of people that worked for him...really to keep on top of all the contractors, and they had effective control over all the activities in that city. But unless you have

A. (cont'd.) somebody with that responsibility, who takes it seriously and learns all the nuances of the activities, you can have the problems, all the problems that you describe.

Q. We have some problem here because the Ministry of Labour would only be notified of such a project if it was over fifty thousand dollars, and I suspect there are a lot of projects this summer going on at forty-nine thousand dollars.

A. Yes, I would say that..we did four projects for fifty thousand dollars in this survey.

Q. Right. So that the Ministry who is responsible for this may not even be notified in most of these projects.

Okay. I would just like to have two more short things that I would like to explore, and one of them is compensation.

Given all of the evidence that you have presented to us over the last two days and in your writings, I think we are terribly concerned about the fact that the Compensation Board in Ontario sets fairly stringent requirements that have to be fulfilled before workers receive compensation, and I guess the requirement that we are most concerned about... first of all there is, of course, the requirement that you be diagnosed as having the disease stated, and that's fair enough. Then secondly, there is a requirement of latency.

But of more concern to us, however, has been the requirement that there be at least ten years of continuous and repetitive exposure before a worker would receive compensation for either mesothelioma, for gastrointestinal cancer, lung cancer or laryngeal cancer, and it seems to me...and I would like you to comment...is that a fair criteria on which compensation should be judged?

A. No, I don't think it is. I think the evidence that disease comes with much shorter exposure is very clear. Nevertheless, it's the situation that with lesser

A. (cont'd.) exposure you have lesser risk, and so somebody...for example, let's take insulation workers. I mean, somebody that worked for five years as an insulator and stopped, and developed a lung cancer thirty years later, might have an exposure such that the probability that that lung cancer was job-related was fifty-fifty. That is, that exposure would contribute to a twofold risk.

Whereas that of somebody working thirty years would have the probability being four to one. He would have a fivefold increased risk.

But there is no way that one can say that there is no risk for less than ten years, or five years, or anything. In the case of mesothelioma, in particular, there is very little evidence that any other exposures would be contributory to that risk, and thus the asbestos exposure per se would be sufficient, irrespective of its length. With lung cancer we are clearly talking about competitive risks.

Q. Right.

A. But that's a very onerous time requirement.

Q. Similarly the latency period...I mean there are a number of requirements on the latency period that are fairly stringently...in some cases fairly stringently applied. You know, the Board always indicates that they give the benefit of the doubt on the individual case, but for instance for stomach cancer, the twenty year latency is being stringently applied. I mean, is that a fair application?

A. No, it is not, again. Again, with insulation workers and the data that I was showing here, we saw that there was an attributable risk to the asbestos exposure, appearing well before twenty years. In most cases it appears afterwards, there is no question about that. But there certainly are cases ...and that attributable risk is also seen for gastrointestinal cancer as well, so that, as I would again emphasize, the latency

A. (cont'd.) period is a statistical phenomenon. There are more cases coming later because of other factors.

But cases that appear earlier than twenty years, some of the cases that appear earlier than twenty years are indeed related to an asbestos exposure in the insulation worker group and, correspondingly, in other groups that we have looked at.

Q. The last question I have, and that was that Dr. Enterline came before this Commission and presented a paper on the proportion of...it was his criticism of the HEW estimates on deaths contributed by asbestos in the U.S., the proportion of cancer due to exposure to asbestos, and it was presented at the Banbury Conference, and I understand that you were there?

A. Yes, that's correct.

Q. Now, I think the one thing that I would like to ask you, is it fair to use asbestosis as the marker disease on which to base these estimates?

A. No, it is not because it reflects on disease prevalence largely in those individuals that would have had the high exposures, and it's appropriate for making estimates in those groups. But in fact, the estimates, the disease experience that will come in future years, from past asbestos exposure, is largely going to be in groups that would have very little, that would have insufficient exposures to create a significant risk of asbestosis, but a sufficient exposure to create a risk of mesothelioma and lung cancer.

Just parenthetically, at that meeting I had presented estimates as well as to the potential risk, based upon other data, that were about twice or maybe a little bit more than twice that of Dr. Enterline, and there we might never agree because one of these subjective things.

But going back to this other issue on that,

A. (cont'd.) I'm sure with discussion Phil and I would agree.

MISS JOLLEY: Thank you very much, Dr. Nicholson.

DR. DUPRE: We're down to the Commissioners' Quarter Hour. Do you want to go ahead, Mr. McNamee?

MR. McNAMEE: I usually have very few questions to ask because I can't think of too many intelligent ones, but I thought I would ask a few questions without intruding too much on the Commissioners' time.

CROSS-EXAMINATION BY MR. McNAMEE

Q. In your study in tab number four, you did a study of certain public buildings in which asbestos had been applied after the year 1958 and up to 1973, is that correct? Some New York buildings?

A. Yes, right. That's the fireproofing use.

Q. I was just wondering, you also did a study of the New Jersey schools in which apparently two hundred and forty-five schools reported the presence of asbestos, and you examined forty-eight. Did you do a similar analysis of when that asbestos had been applied to the schools? I didn't notice it in your studies at all.

A. No, it wasn't reported. We have the information on when it was applied, and some of it could have been applied many, many years...I'm sorry...decades previously.

Q. Yes.

A. Because the use of asbestos for acoustical purposes predated its use for fireproofing...that is, the one being dependent upon the Underwriter Laboratories' approval.

Q. That was my point, that some of this asbestos has been in the schools for maybe...even pre-World War II?

A. Not so much, but it could have been. But it largely was a post-war phenomenon, but it could...some of the schools it did indeed predate 1958.

Q. Without getting into the actual proportion, I guess that you could go back and maybe add that to your study, when the installation...when the asbestos was installed?

A. I perhaps could, but in many cases the people in the school, that were interviewed, were unaware of it. That is, you had turnover of maintenance people, principals, and we didn't ask the school board to go through their records and spend an awful lot of time searching that out, because that would have discouraged their participation in the study.

Q. Well, assuming even in Ontario some of this has been in for thirty years, and in one of your further studies about schools I think you mentioned that maybe two million to six million children may have had, in the United States, some exposure to asbestos as a result of the school system?

A. I think that estimate was of current pupils.

Q. Current pupils? Quite apart from past?

A. Right.

Q. And a hundred thousand to three hundred thousand teachers?

A. Right.

Q. Would it be fair to state that a lot of children would have several years of exposure to an asbestos environment, at least some asbestos environment, in the periods thirty to forty...say thirty years ago?

A. I don't know how many would have had that, but some could have.

Q. Well, wouldn't it be possible...or I don't know whether it has been considered...it would appear to be that in a retrospective basis...I mean, there is speculation that this presence of asbestos in schools may be causing or will cause excess

Q. (cont'd.) deaths of cancer, mesothelioma and other diseases. I just thought on the basis of that...of course, I'm no expert, that there might be some retrospective studies done to check, and one I suggest is that, say if you took the mesothelioma deaths in the United States, say in 1980, and characterized it by age groups and then took...it seems to me that a child exposed at five years old, if the latency period is thirty years, would you be getting deaths that may very well track back to school environment?

A. Yes, the possibility exists of doing that and, as you say, focussing, for example, on women dying between the ages of thirty, perhaps, and fifty, and investigating the school situation that, their possible exposure background as well as the school situation. It's a very difficult study to do. I'm not sure whether the numbers would be sufficient to allow an identification to the extent that asbestos, particularly damaged asbestos surfaces, existing in that period of time would reflect a possible causal relationship to disease.

Q. You mention the difficulty, and I know it would be extremely difficult to take the...just to go by all the school children...and retrospectively something could be done. I'm just wondering, there is this tremendous amount of money being spent in Ontario, and that suggests little documented evidence of any excess disease caused by asbestos in the schools. I don't know whether any studies have been done to verify the prediction...I don't know whether prediction is the...the Ontario Federation of Labour is, of course, saying remove it all. I mean, they don't care what the cost is. That's their standard procedure for anything.

But anyway, I'm just saying, you, yourself, consider that removal is perhaps the best way to cure, shall I say a perceived problem? I mean, if you...

A. A perceived problem, yes.

Q. Is that right? It has to...but you aren't saying remove all asbestos, whether there is a perceived problem or not, is that correct?

A. If there is material on, say you have cementitious or compactible material in a building that has never been affected, is not easily accessible to kids and is clearly presenting no problem, the priority for dealing with that is pretty low.

Q. You are advocating a common sense approach?

A. That's right.

Q. Have you heard of the, I believe it's the Califano Report?

A. I've heard of several. Not specifically, but...

Q. This has something to do...it has been commented upon by Dr. Paul Kotin. Do you know him?

A. Yes.

Q. Does that help you to identify what report he is talking about? It has to do with prediction of excess cancer deaths.

A. Oh, yes. Yes. He also made some comments about schools. So I didn't know whether you were pursuing... Califano did...I didn't know...

Q. Well, Dr. Kotin indicated that the quality of some of the research had been severely questioned by the academic committee, some of the Califano Report findings. Do you have any criticism of Califano, or agree...on the Califano Report? Do you agree or otherwise?

A. The Califano...actually he issued it. It was done by individuals from three institutes of health in the United States. What they did was attempt to look at what might be possible mortality related from past exposure to carcinogens, and in doing so they utilized the limited data

A. (cont'd.) that was available to them in many circumstances, for the workers exposed. This was the National Occupational...I forget what it was called. It was a survey conducted by NIOSH in which individuals went into a one percent sample of plants in the United States to evaluate exposures to different carcinogens, and they had a list that they looked at.

They noted considerable exposures, but in the NOSH...what's called the NOSH report and I forget what the acronym stands for...they did not evaluate the degree of exposure. The HEW...the three institutes report, the Califano report you are speaking of, made some very subjective estimates of the degree of exposure, which I think were excessive in some circumstances, in particular when the estimates I have made based upon other estimates of exposures in worker groups would have fewer individuals deceased in future years from asbestos, as did that report.

Q. I believe you have indicated in one of your studies, and I can't point it out, that the setting of a threshold limit value might, ten, twenty, thirty years down the road people might even criticize that and once the mortality experience and the disease experience has been catalogued, say thirty years down the line, we might even criticize a threshold value that...a low threshold value...that might be accepted today. Is that correct?

A. Well, I think...we see the, in fact even in the last few years, that criticism has arisen from numerous sources of the TLV of two, which has just been recently put forth.

Q. Yes.

A. So the answer is, very definitely that's likely.

Q. Would it be fair to state that the present state of laboratory analysis and predictability, that we can't

Q. (cont'd.) just isolate this in a lab and say well, we can now predict so many deaths at, say point one? There is no way to do it without waiting for the actual event to happen?

A. No, there is. You can make predictions of that in extrapolations from the available data. They are highly uncertain, but it provides guidance as to what your...and you can put limits on those predictions, so you can see what the range of possibilities are from the available data and use that information for regulatory purposes. So I think it's important to use all the information that is available, and not say we are going to take a guess at a standard and wait for thirty years to see if we are right or not.

That has been done in the past, and the history is a very dismal one.

Q. Again, I hope I don't misquote the Ontario Federation of Labour position, but they have taken a position that I think could be simply characterized as - tear out the asbestos, have the government decide that there is, with independent scientists, a safe substitute that will be guaranteed to be nondisease-causing for the next fifty years, and then install it. Is there any...does that seem to you to be...

MISS JOLLEY: Excuse me. That isn't representing what we consider...

MR. McNAMEE: Q. Well, perhaps I'll rephrase that. Assuming we take out asbestos, would you, yourself, know of any...let's say from a fire retardant basis, would you, yourself, know of any materials that are, say, economically feasible to install that we could then say well, this is a material that has a laboratory guarantee of nondisease production for the next thirty years?

THE WITNESS: A. I don't think we can make a guarantee of nondisease production. We can say that some of...I

A. (cont'd.) know that some of the materials that are being used now and are available on the market, economically have far, far less disease potential than does asbestos, comparable asbestos materials.

Q. But could you, yourself, say well, I can guarantee this material...

A. No.

Q. It's perhaps beyond the bounds of present human competence?

A. To a large extent such absolute statements would be, and I don't think anybody would make such absolute statements.

MR. McNAMEE: I have no further questions. Thank you very much.

DR. DUPRE: You are welcome, Mr. McNamee.

I think I would like to point out at this time that this is a good example of the Commission, of course, always being willing to accommodate counsel for maximum amount of time, but we did try very hard at the end of the afternoon yesterday to establish a schedule and I guess...

MR. McNAMEE: Am I being criticized?

DR. DUPRE: ...you or your substitute, Mr. McNamee, were not here. We could have sat, you see, until about six last night, but at five-fifteen...

MR. McNAMEE: Well, I spent five minutes in questioning. I knew that...

DR. DUPRE: Well, we did think we had a schedule, last evening. So all I'm saying, Mr. McNamee, this is not a criticism, I'm just trying to point out how useful it is for parties to try to have someone representing them at the time when we are trying to set the schedule for the second day.

MR. McNAMEE: Yes. Well I have always indicated...I have been selected for selective criticism. The

MR. McNAMEE: (cont'd.) other people put it up to one o'clock.

DR. DUPRE: Well, Mr. McNamee, I just ask you to take that point for what it's worth.

MR. McNAMEE: Thank you.

DR. DUPRE: I'll also ask you, perhaps, to take another...perhaps again this was when you were absent, but the other day we had a lively little moment in the classroom. As the chairman I did issue an invitation to all parties to please refrain from editorializing when asking questions.

Mr. Ublanski, did you wish to ask questions?

MR. UBLANSKI: No.

DR. DUPRE: Dr. Uffen I know has passed. Dr. Mustard, please?

DR. MUSTARD: I have to ask a question, Mr. Chairman, because there is interesting information presented over the past day and a half. On page fourteen of what I guess became tab number...the age at which you receive your exposure to asbestos and the time of onset of cancer, which is the older you are, the greater the latency period will be if you are exposed to asbestos, for lung cancer, but not with mesothelioma.

But then you come to gastrointestinal cancer, which is figure nine of that particular document, in which you show the relative risk does not appear to increase once you've had the exposure. It seems to run fairly...

THE WITNESS: That is added exposure does not appear to give rise to increasing risk, as is the case with the lung cancer.

DR. MUSTARD: That's right. You posed an interesting hypothesis on page fourteen, which gets us back to defence mechanisms and macrophages and everything else, but the point that you make which is an interesting one is, that of course the fibers passing into the gastrointestinal tract

DR. MUSTARD: (cont'd.) are into a different kind of physiological, biological system than fibers going into the lung, and the possibility of their clearance in the gastrointestinal system in fact is...I don't know if any studies have been done, you might not be able to answer this...might be better than in the respiratory tract. That's a speculation, I think, that you put forward...

THE WITNESS: Right.

DR. MUSTARD: ...on the cells. That, of course, would also hold true for the tissue around the larynx, because that's the way they are as well. My question really is, aside from getting into biological implications, has anybody done any studies looking at the risk of gastrointestinal cancer, if it changes with time once you withdraw from exposure?

We were given evidence in one of the earlier hearings that in terms of lung cancer when you pull people out, the risk tends to keep on going for awhile. But if your speculation is correct about gastrointestinal cancer and cancer of the larynx, I am putting in because that's part of the GI system, you might expect if you took people out of the workplace then, that the risk of gastrointestinal cancer might fall. Is there any evidence on that?

THE WITNESS: I looked at the time course in the Patterson group, where they were reviewed, and there tended to be a falloff in recent years, but the numbers were so small that it was hard to make any projections as to what in fact the time course was there.

DR. MUSTARD: In other words, there really isn't any data developed in any of the studies upon this particular...

THE WITNESS: None of a definitive nature. I was just trying to find out...I don't have the...maybe Selikoff. Just a minute.

DR. MUSTARD: While you are looking for it, I

DR. MUSTARD: (cont'd.) just might comment further on it. It would appear the problem in looking at statistics, if indeed that kind of effect is occurring, because depending on how you define the cohort and follow it when you are looking for cancer of the gastrointestinal tract...that's why I picked up the point and was wondering if there is any data that would allow us to have some feel for it, and whether in this particular case if your speculation is correct that the actual risk does decrease after you withdraw people from eposure.

THE WITNESS: Going through all these tables, I can't find anything in here for that experience.

I guess...I'm sorry, I couldn't find a table on the overall...wait a minute, I've got some information...on the overall risk of death at these sites individually. The only number I have is that the gastrointestinal cancer expected, and this would be esophagus, stomach, colon and rectum, it would not include larynx, was twenty-two point seven expected, versus twenty-eight observed. So it's a considerably lower number than is seen in insulation workers where the exposure continues, commensurate with...

DR. MUSTARD: What workers are these?

THE WITNESS: These are the Patterson amosite plant which, of course...

DR. MUSTARD: That had an exposure?

THE WITNESS: That had the exposure, and then it stopped. So this is the group that one would look to to answer your question. Then in looking at that data individually first by exposure category, there was not that much dose-response that would ascribe increasing risk with increasing exposure, again as was seen in the insulation workers because of this factor, and when you look at it with time, I remember that there would appear to be a falloff in older ages, but it wasn't neatly a dose-response

THE WITNESS: (cont'd.) curve that went down, it was just a few scant numbers bouncing around. We are talking about five excess cancers, and that's not easily...not a data set that are easily used to provide definitive information.

DR. DUPRE: Dr. Nicholson, I'll just pose one last question briefly, if I can.

Can we go back to tab nine and look once again at table one?

THE WITNESS: Okay.

DR. DUPRE: First of all, the difference between the best evidence and death certificates with respect to asbestosis, what accounts for the rise in number? Is it, for example, largely cases of heart failure?

THE WITNESS: Yes, there would be a lot of right side heart failure included.

DR. DUPRE: Right side heart failure would be probably the main one?

THE WITNESS: Yes. That would be the principal medical finding that Selikoff would attribute to asbestosis, and in this death certificate categorization it would have to be said as the underlying cause of death, asbestosis.

DR. DUPRE: Could I ask another question? Is it fair to assume that any of a number of the one thousand and... what are we looking at here...no, is it fair, first of all, to assume that any of a number of the two thousand, two hundred and seventy-one deaths may have occurred to individuals who had been diagnosed as being asbestotics?

THE WITNESS: Sorry...is it fair to say?

DR. DUPRE: You have two thousand, two hundred and seventy-one deaths in all.

THE WITNESS: Right.

DR. DUPRE: Now, is it fair to assume that any of a number of those deaths could have been the deaths of

DR. DUPRE: (cont'd.) individuals who simply suffered from asbestosis?

THE WITNESS: Yes.

DR. DUPRE: Do you know how many? How many of all of these cancer deaths, for example, that are recorded here, let us say also had asbestosis or were suffering from it?

THE WITNESS: In part it depends on how you define asbestosis as a disease, because that's not a clear cut entity. I mean you would...and I don't know...the number would depend on that diagnosis. There has to be a certain pulmonary function deficit coupled with a certain x-ray reading, coupled with perceived, with evident shortness of breath. There would be many, but I don't know the number, that would have died of other causes that would, in anybody's criteria, be classified as asbestotic. The number would, of course, depend upon the criteria.

DR. DUPRE: But the raw data in the study wouldn't reveal that?

THE WITNESS: No, because for many of these individuals there were not available data on their pulmonary function prior to death, or on the x-ray evidence of pulmonary scarring.

DR. DUPRE: Just one other point, I appreciate that you have a very, very considerable medical team at Mount Sinai. Would there be a member of that medical team who you would view as particularly expert on the treatment of asbestotics and on what happens to them, what kind of mortality experience they have, the extent to which they may or may not have adverse responses to different kinds of treatment?

THE WITNESS: Selikoff would be the one that would have that...that would be the only one that would have. That is, in that he has the clinical data that he has collected on the New York and New Jersey group, and corresponding mortality

THE WITNESS: (cont'd.) data on people that he had examined at different periods of time.

So in terms of the future mortality according to clinical signs at a point in time, that would be best be answered by that study. We have a very limited bit of information in that only about twenty or thirty people have died, of those in the Bedford group that we have followed up with the mortality related to the clinical findings in 1974. But it's twenty or twenty-five people, I recall.

So that's the second set that we have that would... and that will in fact be published before too long, I believe.

DR. DUPRE: This will show what, again?

THE WITNESS: It would look at the causes of death, see if the causes of death...sorry, let me say that another way...to see if the manifestation of clinical signs of asbestosis are predictive of greater or lesser mortality in future years, and to what degree they are.

DR. DUPRE: I guess, you see, quite aside from predictability, I have an interest in terms of compensation policies, simply in terms of, you know, what has happened to individuals who were asbestotics, what they died of other than right side heart failure, who might have expertise in that... the kind of nexus that exists, let us say, between asbestosis and various causes of death.

THE WITNESS: Yes, there may be some information, and I don't have it because I haven't been with that study, on the background, the deaths of people who had received compensation for asbestosis, in the United States. Selikoff had a study, and Peter Barth, I understand, as part of the review process that you are undergoing, and he was a participant of that.

DR. DUPRE: He is doing a study for us.

THE WITNESS: That information may be available from him. I don't know the extent to which it is.

DR. DUPRE: I see. So the study that you are thinking of there would be what has been called the Barth/Selikoff study that was done for the insulators, the co-operation of the insulators...?

THE WITNESS: Yes, the compensation experience of insulation workers in different parts of the country, as well as three different groups of asbestos-exposed individuals in New Jersey, were looked at. So we started with a mortality cohort and went back and asked what compensation experience they had. It's retrospective in that sense, but it may provide some of the information you are seeking. But I don't know the details enough to discuss it.

DR. DUPRE: Miss Jolley, would you care to rephrase my questions in any way, on this subject?

MISS JOLLEY: No, I just...

DR. DUPRE: Okay.

Well, please, may I, on behalf of all of us, Dr. Nicholson, thank you most warmly indeed for your generosity with us and for having spent the time. Thank you very much.

THE WITNESS: Thank you.

DR. DUPRE: We'll now adjourn until Tuesday, the 7th of July...is that correct...at ten a.m.

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THE INQUIRY ADJOURNED

THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

EDWINA MACHT